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THE MALIGNANT NATURE OF BRONCHIAL ADENOMA

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WESSLER and Rabin,[1] in 1932, reported the separation of bronchial adenoma from bronchiogenic carcinoma, and they pointed out that some of these tumors became malignant. By 1941 we[2] had reported eighteen cases of bronchial adenoma and stated that these tumors exhibited growth potentials midway between distantly metastasizing carcinomatous and purely local polypoid bronchial tumors. In these publications clinical and pathologic manifestations were discussed.[3, 4] Treatment was concluded to include bronchoscopic removal in certain instances but sooner or later because of the growth of the extrabronchial portion or suppuration in the distal lung, pulmonary resection was indicated. Subsequently a personal experience with approximately fifteen additional cases has confirmed the previously reported findings. However, during the past few years the publications by Womack and Graham,[5] Anderson,[6] Chamberlain,[7] and Crafoord[8] have served to emphasize the potential malignant qualities inherent in these tumors. In 1947 I[9] published follow-up results of operated cases.[6] Six of these ten patients had survived more than five years since operation without appearance of any distant metastases or spread. By now, two additional patients of this group have passed the five-year mark, giving 80 per cent so-called five-year cures.

It was always stated that this subject although greatly clarified is still somewhat obscure, because some varieties of bronchial adenoma may be more malignant than others (Brunn[10]). The recent report of the Army Institute of Pathology[11] has shed some light on the question of varieties of bronchial adenomas. A histologic division into predominantly carcinoid and mixed tumor type has been described, and the mixed tumor type is probably more apt to exhibit greater degrees of malignancy.

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During the past two years observations have been made upon an additional unreported group of patients which tend to indicate that a high degree of malignancy may be exhibited by bronchial adenoma, especially of the mixed tumor type, until at post-mortem bronchiogenic carcinoma rather than mixed tumor type of bronchial adenoma is diagnosed. Womack and Graham[5] had indicated that such a malignant transformation from so-called benign bronchial adenoma could occur. This first case to be reported is the only one of approximately forty cases where such a high degree of malignancy did occur and is one of two cases observed, both of the mixed tumor type and both of which ended in death. In each, endoscopic and radiation treatment without pulmonary resection resulted in recurrence of the tumor located at the bifurcation of the trachea.

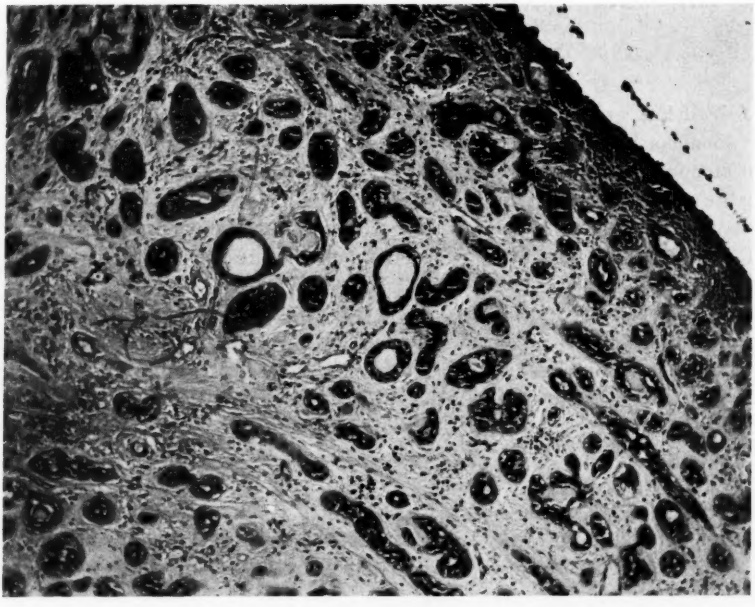

Fig. 1 (Case 1).—Bronchoscopic biopsy April 14, 1942, E. S. Note the mixed tumor pattern from the tumor of the lower end of the trachea. (Microphotograph ×85.)

CASE 1.—E. S. (DA-22-48), a white man, aged 58 years at the time of death, first entered the Cedars of Lebanon Hospital April 14, 1942, with complaints of asthma, chronic bronchitis, and hemoptysis. Bronchoscopy was done on this date and a biopsy was taken from a nickel-sized polypoid tumor at the lower end of the left wall of the trachea which occluded the trachea to approximately one-fifth of normal. The bronchoscopic biopsy was reported as bronchial adenoma. Fig. 1 shows this biopsy, and the fact that it conforms to the mixed tumor type of bronchial adenoma was confirmed. During the next year several bronchoscopic examinations were made with cauterization; radon and x-ray therapy were administered. Subsequent bronchoscopic examinations up to January, 1944, disclosed only little tumor remaining. Dyspnea became progressively worse in 1945 and 1946, requiring several emergency bronchoscopies at which time very little tumor tissue was noted but the left stem

bronchus was narrowed to 50 per cent of normal as well as the trachea, and purulent secretions were aspirated. In 1947 the tumor mass was more prominent and cauterization was again resorted to. However, attacks of tracheal suffocation and severe dyspnea continued. With the idea of performing a left pneumonectomy and local resection of the tumor, exploratory thoracotomy was performed on Nov. 21, 1947. Shortly after the left pleural cavity was entered, the anesthetist announced that he was unable to aerate the patient's lungs through the intratracheal tube. The lower end of the trachea was rapidly exposed and an incision was made in the anterior wall of the last centimeter of the trachea. Through this a No. 20 French catheter was passed into the right stem bronchus and oxygen was administered followed by improvement in the patient's condition. From the center of this transverse tracheotomy another incision was made inferiorly for 3 cm. into the left stem bronchus. The edges of this incision were approximated to the proximal edge of the transverse tracheotomy and the anesthetist announced that better aeration was established. The catheter was then removed and adequate oxygenation was carried on as the result of the "Tee" plastic procedure performed. Additional findings were pleural implants from 1 to 5 mm. in diameter, infiltration of esophagus and trachea, and suppuration of the left lower lobe. The postoperative course was fairly smooth; neither tracheopleural fistula nor empyema developed. He was discharged on the fourteenth postoperative day but returned with acute tracheal obstruction six weeks later, and was relieved by bronchoscopic aspiration only to succumb to another attack after three weeks.

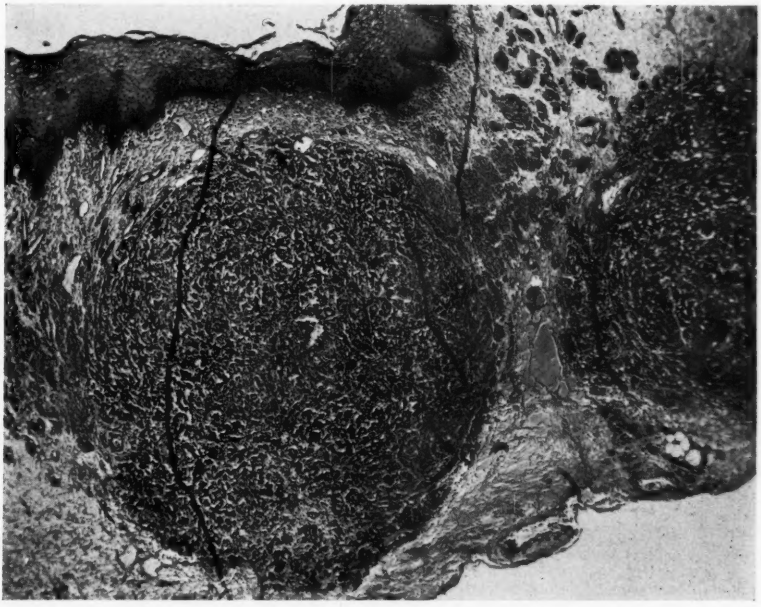

Fig. 2 (Case 1).—Post-mortem specimen, E. S. Metastatic nodule invading esophagus. (Microphotograph ×35.)

Post-mortem examination showed carcinoma of trachea with invasion of the upper trachea, esophagus (Fig. 2), mediastinum, and metastases to pericardium, lungs, left parietal pleura, adjacent lymph nodes, and prevertebral fascia of the neck (Fig. 3). Microscopic examination showed not only the "Swiss cheese" mixed tumor appearance of some areas of the malignant tumor but also areas of frank malignant cells (Fig. 4). There seemed little

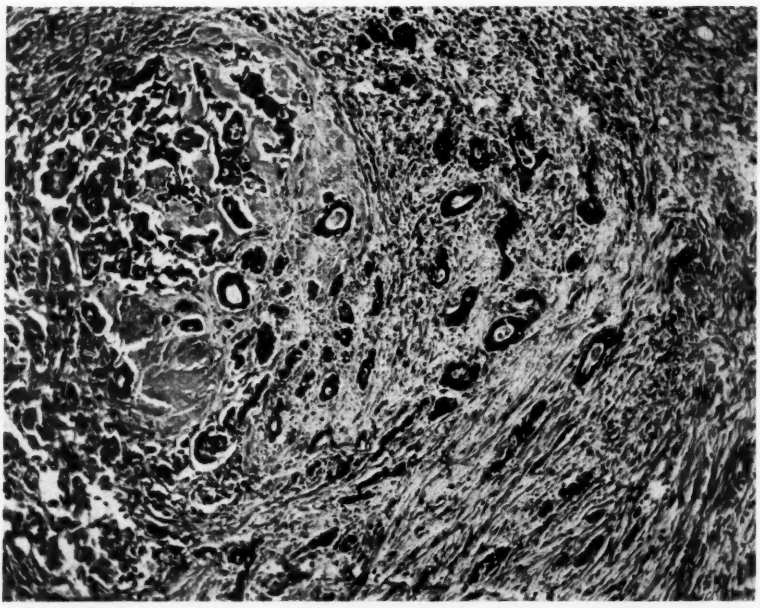

Fig. 3 (Case 1).—Post-mortem specimen, E. S. Note the tendency to reduplicate the mixed tumor pattern seen six years before in the bronchoscopic biopsy. (Microphotograph ×85.)

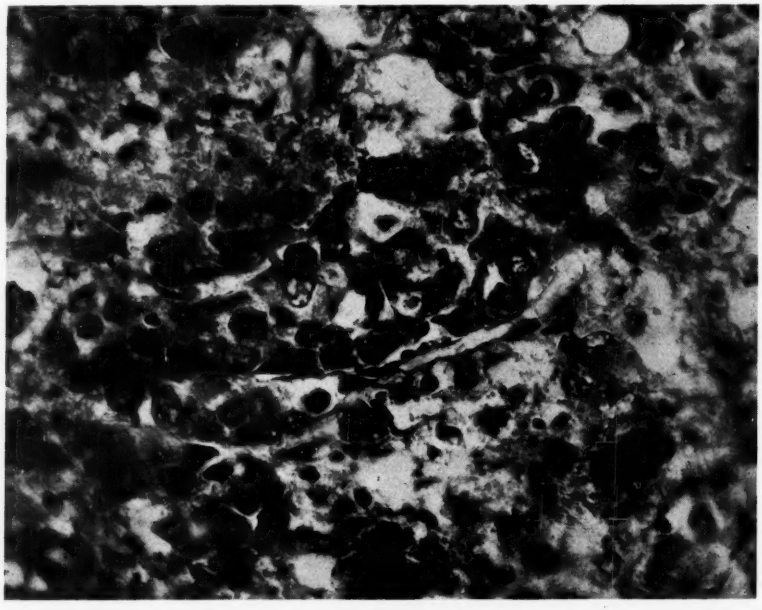

Fig. 4 (Case 1).—Post-mortem specimen, E. S. Note numbers of frank malignant cells with large irregular nuclei. (Microphotograph, ×450.)

doubt that a mixed tumor type of bronchial adenoma had become, in approximately six years of treatment and observation, a highly malignant carcinoma. Nor is there much doubt that if this neoplasm were seen only at post-mortem (without the 1942 biopsy, Fig. 1) it would have been diagnosed as an undifferentiated carcinoma of bronchiogenic origin.

The significance of this case may be that certain bronchiogenic carcinomas, particularly those of long duration[12] had their origin in a bronchial adenoma. It is urged that all bronchial adenomas continue to be observed for malignant transformation.

The following cases illustrate less severe degrees of malignancy, especially invasiveness and local metastases.

Fig. 5 (Case 2).—Surgical specimen, right lower, right middle lobes, K. F. Note the large size of the tumor in spite of the short history.

CASE 2.—K. F. (S-362-48), a white woman aged 30 years, was admitted to the Cedars of Lebanon Hospital, Jan. 25, 1948, referred by Doctor J. M. Krevett. Onset of symptoms was Dec. 1, 1947, with persistent cough, weakness, and pain in the right chest. X-ray films of the chest showed a tumor mass in the right lower lobe. Bronchoscopic examination was performed January 26, 1948, with findings of a bronchial adenoma obstructing the bifurcation of the right stem bronchus at the take-off of the right middle lobe. Right middle and lower lobectomy was performed on Jan. 29, 1948. The tumor was 7 cm. in its greater diameter (Fig. 5), invaded through bronchial walls about the inferior pulmonary vein, and involved a

neighboring lymph node by contiguous spread (Fig. 6). Very little distal bronchiectasis was present. Microscopic examination showed the characteristic "Swiss cheese" (Fig. 7) pattern of the mixed tumor type of bronchial adenoma. The patient was discharged on the tenth postoperative day and has remained symptom-free these past five months.

The microscopic appearance with the invasiveness and large size together with the short duration of symptoms (two months) all indicate a more rapidly growing tumor than is usually expected with the carcinoid type of bronchial

Fig. 6 (Case 2).—Surgical specimen, K. F. Note the invasion through the bronchial wall of adenoma of the mixed type and the invasion of neighboring lymph node by contiguity. (Microphotograph, ×6.)

Fig. 7 (Case 2).—Surgical specimen, K. F. Note the "Swiss cheese" appearance found in the mixed tumor type of bronchial adenoma. (Microphotograph, ×125.)

adenoma. No areas of malignant transformation such as seen in Case 1 were present, but one wonders if this tumor were allowed to remain whether more malignant features would have developed. Also the future might well show that it would have been better to have performed total pneumonectomy instead of right lower and middle lobectomy. It is urged that patients treated with only lobectomy be carefully followed for recurrence and metastases, especially if they are microscopically of the mixed tumor type.

The next two cases are bronchial adenomas of the carcinoid type of extremely long duration and occurring together with neoplasms arising independently in other organs of the body. In both these patients local invasion but not distant metastases occurred.

CASE 3.—L. B. (S-1430-47), a 60-year-old man, entered Birmingham Veteran's Hospital on March 10, 1947, complaining of weakness, fatigue, and cough productive of mucopurulent sputum. He had a history of frequent respiratory infections from 1917 to the time of admission. There was a hemoptysis in 1922 and since that time there had been intermittent subsequent pulmonary hemorrhages, often lasting several days and of a pint or more. He was hospitalized on many occasions between 1922 and 1947 and given the various diagnoses of pneumonia, lung abscess, and empyema. He had the diagnosis of pneumonia on four different occasions. In July, 1933, a right phrenicectomy was performed and two months later a right thoracotomy was done to drain an empyema. Following this, the patient's symptoms were markedly improved but a chronic draining chest wall sinus continued to drain for the next fourteen years. Communication with the bronchial tree was established and chronic lung abscess with bronchocutaneous fistula was diagnosed.

Chronic lymphatic leucemia was diagnosed in 1943 at the San Diego Naval Hospital. White blood counts as high as 160,000 per cubic millimeter were reported. Fatigue and lassitude were the main symptoms on entry.

Past History.—There had been no serious diseases.

Family History.—There was no history of tumor or cancer in the family but the patient's mother had died in middle age of what possibly was pulmonary tuberculosis.

Examination.—Physical examination revealed a thin, pale, chronically ill appearing 60-year-old white man. There was an old healed 10 cm. thoracotomy scar in the right posterior axillary line. A small "pencil-point" draining sinus was present in the seventh interspace. There were fine moist râles in the right apex and dullness over the entire right lower and middle lobes. A permanent right diaphragmatic paralysis was present.

Laboratory Findings.—X-ray views of the chest showed elevation of the right diaphragm, regenerated eighth rib, deviation of the trachea to the right, and probable shrinkage of the right lower and middle lobes. Subsequent x-ray films and planigrams demonstrated a large mass in the paramediastinal area of the right lower part of the chest. Examination of the blood revealed normal red count and hemoglobin, white blood count 66,300, 71 per cent lymphocytes, 27 polymorphonuclear leucocytes, and 2 monocytes. Bone marrow puncture of the sternum, March 27, 1947, showed lymphatic hyperplasia of the bone marrow characteristic of subacute lymphatic leucemia and many pro-lymphocytes were present.

Bronchoscopy was done on the patient for the first time March 19, 1947, which revealed a bosselated mass in the right main stem bronchus 1 cm. below the carina, completely occluding, and covered with a grayish removable exudate. First biopsies showed only necrotic tissue. One week later bronchial adenoma with extensive necrosis was diagnosed from the biopsy. Bronchial smears made at this bronchoscopy were positive for tubercle bacilli.

Pyuria and hematuria developed in April and cystoscopically a transitional cell carcinoma of the bladder was fulgurated and removed. Repeat bronchoscopy July 22 showed no essential change. More tumor was removed for study. On Sept. 12, 1947, a transurethral

resection of the prostate was performed for obstructive uropathy. Microscopic examination of the prostatic tissue was benign prostatic hyperplasia. An acute tuberculous pneumonitis appeared in the previously clear right upper lobe at this time.

The bladder carcinoma was now under control and the patient was prepared for a right pneumonectomy with the aid of streptomycin. On Oct. 15, 1947, a right pneumonectomy was performed. Surgical specimen showed a large tumor, not only in the right stem bronchus, but also invading toward the mediastinum. There were several enlarged lymph nodes in the mediastinum from 1 to 3 cm. in size. One of these showed a fairly acute tuberculous process. The postoperative course was relatively uneventful. One month following the pneumonectomy a partial thoracoplasty was performed to protect against overexpansion of the left lung. Recovery following this operation was uneventful.

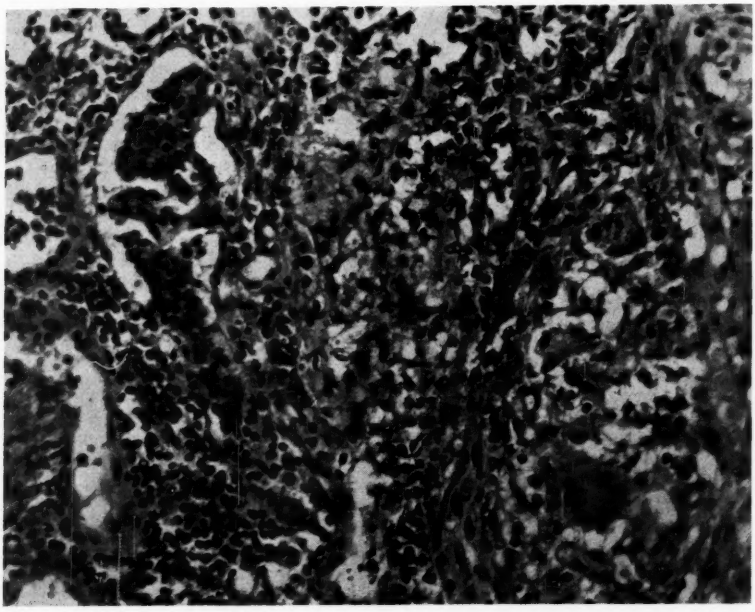

Fig. 8 (Case 3).—Surgical specimen, L. B. Note the Langhans' giant cells and the adjacent invading bronchial adenoma. Bronchial adenoma invading tuberculous lymph node. (Microphotograph, ×200.)

The measurements of the tumor were 3.75 by 6.25 cm. Large blood vessels were compressed by the tumor. Several acute tuberculous nodules were seen close to the pleura on cut section of the lung. In one area the tumor invaded by contiguity a tuberculous lymph node (Fig. 8). Microscopic sections of the tumor showed the pattern to be consistent with carcinoid type of adenoma. Another lymph node showed a clump of tumor cells attached to the capsule, and a third lymph node showed small masses of tumor cells in the capsule and invading the parenchyma of the lymph node. A diagnosis was bronchial adenoma with metastases to surrounding lymph nodes and fibrocaseous tuberculosis, with acute dissemination.

Five months after the thoracoplasty a series of 20 negative sputum smears and cultures had been obtained, and 110 days of streptomycin treatment had been completed. The patient was discharged home with maximum hospital benefits. At the time of discharge the white blood count was 73,000 but it had dropped as low as 15,000. Cystoscopies showed no further evidence of the carcinoma of the bladder.

This case illustrates the malignant nature of the carcinoid type of bronchial adenoma that is associated with pulmonary suppuration for approximately thirty years. Only local invasion of mediastinum lymph nodes has occurred. The interesting features of concomitant presence of chronic lymphatic leucemia, transitional carcinoma of the bladder, and pulmonary tuberculosis are difficult to tie up in any clinical pattern. These findings together with a bronchial adenoma suggest that the origin of a bronchial adenoma may occur from the same stimuli that produce tumors in general in the body. It is of still greater interest that all of the three neoplastic disturbances in this one patient were relatively benign, in spite of the microscopic malignancy. It would appear that the carcinoid form of bronchial adenoma takes on a much more benign course clinically than the more malignant mixed tumor form of bronchial adenoma.

CASE 4.—L. S. (DA-4-48), a white woman 46 years of age at the time of death, was admitted to the Cedars of Lebanon Hospital Dec. 30, 1947. Thirty years before entry she had had a right empyema which was drained and ever since that time had a persistent draining sinus of the chest wall. Bronchoscopies had been performed as long ago as twenty years and a lesion in the right stem bronchus had been described. Approximately ten years before, a positive biopsy for a bronchial adenoma was obtained. General condition though not good allowed her to carry on a fairly normal existence until 1946 when she began to develop symptoms of an intracranial lesion. A pituitary adenoma was later diagnosed, and when she entered on this admission, she suffered from severe headaches, faintness, visual blurring, staggering and instability of equilibrium, as well as dyspnea, orthopnea, and palpitation. On physical examination she was found to have coarse skin, large tongue, hypertrichosis, absence of breath sounds in the right base, with râles on cough. Blood pressure showed a mild hypertension. X-ray therapy had been given one year before to the pituitary, and on entry x-ray examination of the skull showed erosion and enlargement of the sella turcica. Following entry, her condition remained poor and became critical one week after entry, she died Jan. 6, 1948.

The post-mortem examination showed 2 cm. from the main stem bronchus on the right a large adenoma which measured approximately 3 cm. in its greatest diameter. There were two large cystic cavities distal to the obstruction caused by the adenoma. These were approximately 4 cm. in diameter. The bronchial adenoma invaded through the wall of the right stem bronchus and the extrabronchial portion measured 7 cm. in greatest diameter. The fistulous tract communicated with the bronchi and the skin of the thoracoplasty scar. There were several enlarged mediastinal lymph nodes. There were bilateral adenomas of the adrenal glands, cor pulmonale, mixed adenomas of the pituitary gland with proliferation of all three cell types, early malignant transformation, and persistent large pineal gland. Microscopically, the tumor was of the carcinoid type and some granules in the cytoplasm could be demonstrated with argentaffin staining. Invasion of the mediastinal lymph nodes and the adventitia of the pulmonary artery were demonstrated, and in another section a dumbbell mushrooming of the adenoma into a large pulmonary vein was noted (Fig. 9).

This case demonstrates vascular invasion, as well as mediastinal lymph node invasion, in a carcinoid type of bronchial adenoma of over thirty years' proved duration. The concomitant adenomas of the adrenals and pituitary gland suggest again that whatever stimulus produced the bronchial adenoma stimulated other organs to produce adenomatous growths in them. Again, the low-grade invasion of the carcinoid type of bronchial adenoma is demonstrated. In spite of definite evidence of invasion into the lumen of a large pulmonary

Fig. 9 (Case 4).—Post-mortem specimen, L. S. Note the mushrooming of the bronchial adenoma through the vein wall. In spite of tumor floating in the lumen of the vein no vascular seeding of bronchial adenoma could be found. (Microphotograph ×6.)

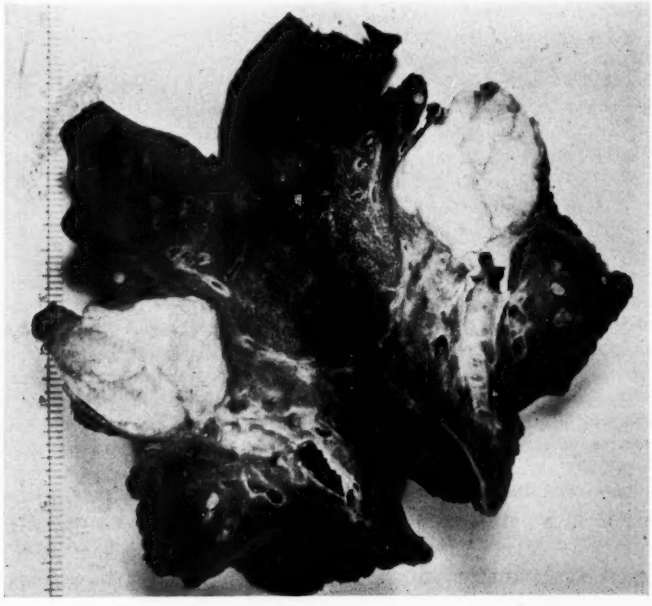

Fig. 10 (Case 5).—Surgical specimen left lower lobe, A. K. Note the metastatic nodule in the lymph node close to the large adenoma.

vein, no metastatic vascular seeding was demonstrated. This suggests that the environment favoring growth of bronchial adenomas of this type is found in the bronchus and not elsewhere in the body. The relatively small growth found in the metastatic lymph nodes also favors this point of view. Adenoma which invades lymph nodes has not been observed to reach the proportions commonly found with bronchiogenic carcinoma when it invades lymph nodes.

CASE 5.—A. K. (Registry No. 2103), a white man, aged 25 years, first entered the Birmingham Veteran's Hospital Aug. 23, 1946. Onset of symptoms was two months before, with a pneumonia. Bronchoscopy was first performed October 3 with the findings of a polypoid tumor attached to the medial wall of the left lower lobe bronchus and bronchiectasis. Bronchoscopic biopsy was bronchial adenoma, carcinoid type. X-ray films confirmed the presence of a tumor mass. Lobectomy of the left lower lobe was performed Oct. 29, 1946. The specimen removed revealed a large ulcerated adenoma of the bronchus attached at the base of the markedly dilated left stem bronchus completely obstructing it. There was marked invasion of the adenoma through the bronchial wall into the adjacent lung parenchyma and what appeared to be a recent increased rate of growth associated with this extension through the bronchial wall. Two small recent metastases to peribronchial lymph nodes were demonstrated (Fig. 10). Microscopically, the tumor was of the carcinoid type and it is noteworthy that at the base of the tumor where it was attached to the bronchial wall there were a large number of stellate type of cells. The tumor that appeared beyond the bronchial wall was somewhat different although the general pattern was retained. These cells were larger, the cytoplasm more pale-staining, and the nuclei varied distinctly in size, some of them being fairly large and hyperchromatic. Mucicarmine stains revealed no mucous production by the cells. X-ray pictures were later made of all of the bones of the body and no metastatic lesions were seen. Electroencephalogram suggested some abnormality of the cerebral cortex but nothing clinical could substantiate these findings. One year after operation the patient was heard from by mail and was apparently well.

This represented apparently a carcinoid bronchial adenoma with fairly early metastases to regional lymph nodes and a short duration of symptoms. Lobectomy was done although metastatic tumor occurred in the mediastinal lymph nodes of the hilum of the left lower lobe. The follow-up on this patient will be watched with keen interest.

SUMMARY AND DISCUSSION

Five patients showing malignant qualities in bronchial adenomas are reported. The most malignant changes to occur were found in the mixed tumor type of bronchial adenomas. The complete transformation to a clinical and histologic picture of bronchiogenic carcinoma has been observed and is reported herein. It is suggested that carcinoma of bronchiogenic origin may at times arise in a mixed tumor type of bronchial adenoma. Carcinoid types of bronchial adenoma not infrequently show metastatic involvement of the mediastinal lymph nodes. Observations are presented which indicate that this may occur when the tumors have been present for as long as thirty years or when the duration of symptoms is only a few months. The finding of bronchial adenoma in conjunction with multiple tumors of low-grade malignancy in other organs suggests that the stimulus for the production of bronchial adenoma is the same as that capable of producing neoplasms in other organs than the lung. The failure of bronchial adenoma to produce vascular seedings even though the tumor cells were demonstrated floating in the lumen of a large pulmonary vein suggests that the only favorable soil for bronchial adenoma is in the bronchi and lung.

The treatment of bronchial adenomas by lobectomy and pneumonectomy has given approximately 100 per cent five-year cures where this period of time has elapsed since operation. From the observations on tumors of over thirty years' duration it would appear that several decades will be necessary to observe patients treated by lobectomy and pneumonectomy before the recurrence rate can be definitely established. The malignant nature of bronchial adenoma is not so malignant nor is its benign course so benign.

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(See discussion on page 155.)

ADENOMA OF THE BRONCHUS

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THE first report from the Mount Sinai Hospital of the features of a group of cases of bronchial adenoma was made by Wessler and Rabin in 1932. At first there appeared to be general acceptance of the views which were expressed concerning pathology and treatment. In the original paper involvement of the tissues beyond the bronchial wall was described in two cases. Malignancy of bronchial adenoma has been emphasized to such an extent in recent years that the prevailing opinion at the present time appears to be, first, that the tumor cannot be removed completely bronchoscopically and, second, that surgical eradication should comprise the sole treatment as in the case of cancer of the lung. Whereas some regard the lesion to be actually malignant, others believe that radical treatment alone is permissible because of potential malignancy.

Before general consideration of the subject, the microscopic features of bronchial adenoma should be described. The tumor consists of small cells with regular round or oval nuclei and rare, if any, certain mitoses. Nucleoli are inconspicuous. The cells are either cuboidal, cylindrical, or polyhedral and the cytoplasm is usually scanty and may be entirely clear. The cells are generally arranged in groups, separated by a fine stroma, but in places this may be thick and hyalinized. In these locations, the nuclei are often elongated and may be irregular nad pyknotic, suggesting carcinoma. The same irregular and pyknotic appearance may occur at the periphery of the biopsy specimen, where the tissue has been crushed by bronchoscopic forceps and may also cause an error in diagnosis if the regularity of the nuclei in the center of the specimen is not appreciated. True gland formations are rare. However, the occurrence of glandlike formations is not uncommon.

The only two types of carcinoma that may be simulated are adenocarcinoma and small round or oat cell carcinoma. Adenocarcinoma of the bronchus is composed of large cells in contrast to those of bronchial adenoma. The real difficulty lies in the differentiation from small cell carcinoma. The presence of many mitoses, which is characteristic of small round or oat cell carcinoma, will decide the diagnosis in most instances. The difficulty in differentiation from the occasional case of small cell carcinoma with regular nuclei and with rare or absent mitoses can usually be resolved by a study of the arrangement of the cells and their relationship to the stroma. If the cells are arranged in gland formations, the diagnosis of small cell carcinoma may be excluded. Furthermore, the cells in small cell carcinoma are distributed in

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From the Mount Sinai Hospital.

irregular masses without definite arrangement in relationship to the stroma. The mosaic-like alveolar pattern, with groups of cells separated by a fine stroma, is not seen in small cell carcinoma. In the absence of glandlike formations or of a solid alveolar pattern, the arrangement of the cells in the form of double rows or palisades separated by linear fine stroma will also serve to differentiate adenoma from small cell carcinoma. Infiltration or the appearance of infiltration of the stroma has, in the past, led to confusion with carcinoma, but this finding should be disregarded.

Oftentimes there is the possibility of grave error in diagnosis when tiny bronchoscopic specimens are studied microscopically. Sections revealing only squamous or columnar epithelium or granulation tissue containing a few small cells can be misleading. When these are obtained from the region of a polypoid tumor which has been seen bronchoscopically, additional specimens should be removed before venturing a diagnosis either of squamous cell tumor or of granuloma. In cases of adenoma, satisfactory specimens should be available from which the correct diagnosis can be made. According to our experience, errors in the diagnosis of bronchial adenoma are very rarely made if the microscopic features which have been outlined are given consideration and the precautions concerning specimens are borne in mind.

In accordance with the foregoing description, uniformity of the cells and the rarity or absence of mitotic figures proclaim the benign nature of the neoplasm in so far as microscopic features are concerned. There was a period in which pathologists confused adenoma with malignant growths, but this phase has passed and it is safe to say that at the present time most pathologists agree on the distinctive microscopic features of the neoplasm to which the name "adenoma" is generally attached. Now, however, there are those who regard "adenoma" to be a misnomer on the basis of microscopic features, said to be those of mixed tumors or of carcinoma.

Against this background we wish to present an analysis of a series numbering more than 64 cases, because the continued study and follow-up for many years permit conclusions drawn from long observation and experience. Before doing so, however, a survey of the literature on the subject is in order. A complete analysis made in collaboration with Dr. Oscar Friedman will be set forth in another publication; only some features germane to our present purpose will be mentioned here. Excluding cylindromas, at least 368 certified cases of bronchial adenoma were found in the literature. By "certified" we mean cases with specimens removed by bronchoscopy, by operation, or encountered at autopsy. Statements which lack such evidence are omitted for obvious reasons. Of the 368 cases, 100 consisted of operative removal with reported specimens, 23 were autopsies, and the remainder, bronchoscopic material. Significant features in relationship to the question of malignancy can be gleaned only from the operative and autopsied cases. They can be summarized as follows:

1. There were 43 lobectomies without a single instance of involved nodes. Pneumonectomy was performed in 60 cases, of which 13 had involved nodes.

Of the 23 autopsied cases lymph node involvement existed in 5. There were 3 instances of remote metastases: a nodule in the opposite lung, one in the liver, and a microscopic metastasis in the bone marrow without lymph node involvement. In any event, there is no reported case presenting the usual features of malignant extension.

2. It is impossible to ascertain the proportion of cases in which extension beyond the bronchial wall existed, and therefore in which complete eradication endoscopically might have been feasible. Extension beyond the bronchial wall is slight in most of the reported cases. There is no ulceration, necrosis, or diffuse invasion of adjacent tissue when so-called malignant transformation takes place.

3. Involvement of lymph nodes, which in the reported cases is limited, is usually by direct extension from the primary tumor, the involved nodes being immediately adjacent to the tumor.

4. "Malignant transformation" is characterized solely by immediately adjacent involved nodes or remote, usually minute, metastases which have been already mentioned as extremely rare occurrences. In either case there are repeated microscopically the features of the primary tumor, uniformity of cells and, with rare exceptions, absence of mitoses.

5. The mean period of the duration of symptoms in cases with lymph node involvement was from twelve to fifteen years, indicating that the metastases were either of long duration or occurred very late in the disease, in contrast with the usual course of malignant tumors.

6. Whereas the average age of onset of symptoms in bronchial adenoma was 28 years in 100 reported cases, the mean age of onset of patients with metastases was decidedly less (about 20 years). Thus, the literature portrays a most peculiar and benign form of malignancy, encountered in less than 10 per cent of the cases.

It can be stated at once that an analysis of our 64 cases yields results which are not significantly at variance from those we have culled from the literature. However, our long follow-up study offers a rounded picture of the life history of bronchial adenoma which cannot be obtained from the literature. Furthermore, case reports often are lacking also in the details of clinical features which are related so closely to the life history of bronchial adenoma, and some attempt will be made here to repair this ommission.

Turning briefly to a restudy of the sections of the 64 cases we may say that bronchial adenoma is established as a distinctive pathologic entity. True gland formation being absent in most instances, the term "adenoma" is inaccurate to this extent, as also in thyroid adenoma for example. All the characteristics of mixed tumors are not seen. Calcification and ossification as noted in adenomas elsewhere are unusual. Islands of cartilage or bone almost always represent remnants of cartilaginous rings which may be ossified. Cylindromas comprise a separate entity and should not be included with bronchial adenoma.

The microscopic features and macroscopically circumscribed limits of bronchial adenoma were maintained in the 5 instances of metastases in our series. Of these 5 cases the metastasis was confined in 2 cases to a solitary unusually large node immediately adjacent to the smaller primary tumor. The age of one of these patients was 14 years, of the other, 64 years. The roentgen evidence in the former points to the presence of the metastatic node essentially unchanged for a period of more than five years. Thus, as in the literature, the term "malignant transformation" applied to bronchial adenoma is one of common usage but is not justified by the evidence. Instead, each case of lymph node invasion can be properly referred to as a case of bronchial adenoma with regional lymph node involvement. Even in the presence of metastases the term bronchial adenoma should be retained, in our opinion.

A summary of the clinical course in our cases reflects the essentially benign nature of bronchial adenoma. Unless complicated by infection the course is not progressive as in malignant tumors. This is in keeping with slight growth over periods of many years. The course also remains essentially unaltered in the presence of so-called malignant transformation as signalized by involvement of adjacent lymph nodes.

A study of our case histories reveals a variety of presenting clinical pictures. There may be mentioned in passing cases in which the first symptoms and physical signs are those of recurrent pneumonia, bronchiectasis, pulmonary abscess, or stenosis of the middle lobe. However, we wish to emphasize here a syndrome which can be properly termed an "adenoma syndrome" because it was present in at least 32 of 62 completely documented cases. It consists of a history of recurrent clean hemoptysis (fresh blood not admixed with purulent secretion) and evidence of bronchostenosis (by symptoms, physical signs, and roentgenogram), occurring in a young individual. In an older individual the syndrome is distinctive of adenoma when of long duration. There is also an "adenoma syndrome" of a negative sort in which there is evidence of bronchial obstruction, with no sputum and no hemoptysis.

Turning now to the situation of the tumor, we can report on 58 cases in which our information is precise. Using terms which are generally employed, the bronchial adenoma was situated in a main bronchus in 12, upper lobe bronchus in 5, lower lobe bronchus in 26, middle lobe bronchus in 6, and in a branch bronchus in 9. The middle lobe lesion is worthy of separate consideration but for present purposes (that is, discussion of therapy) can be regarded as a branch bronchus. The degree of invasion of the bronchial wall can be stated with certainty only in removed or autopsied specimens of which we can report on 25. In 6 the tumor was purely endobronchial. In most of the remainder, the extension through the bronchus was limited. In 2 the extrabronchial portion was very much larger than the endobronchial. It is proper to point out that vertually all these cases were operative ones, and as such are to be contrasted with those in which bronchoscopic treatment was employed.

The cases in which treatment was bronchoscopy and which will be reported in detail by Kramer and Som are referred to here chiefly because of cures which were achieved. The following results are stated on the basis of long follow-up: Adenoma of main bronchus, 5 cures, 1 failure; of upper lobe bronchi, no cures, 2 failures; of lower lobe bronchi, 7 cures, 7 failures; of middle lobe bronchi, 2 cures, 4 failures; of branch bronchi, no cures, 3 failures. Cases are listed as cures only when repeated biopsies of the base at intervals of months to years after endoscopic removal of the tumor establish the absence of neoplastic tissue. It will be noted that the most favorable area for cure is adenoma of a main bronchus. The reason may be in part more ready accessibility but also a lesser tendency to bronchial penetration of main bronchus adenomas. This is high lighted by a study of the specimen in one case in our series in which pneumonectomy was performed but in which cure by bronchoscopic treatment could certainly have been achieved. It should be emphasized that failure of treatment to effect a cure in our series of cases is not synonymous with failure of treatment since not a few of the patients were cured symptomatically for long periods of time. Furthermore, one of the indications for radical operation as stated in the literature is infection distal to the neoplasm. Undoubtedly bronchiectasis resulting from tumerous bronchostenosis and infection is irreversible but this does not necessarily demand surgical eradication. In our series were 5 patients with suppurative bronchiectasis in whom there was not only complete endoscopic removal of the tumor but also disappearance of infection.

There is not sufficient time for a discussion of the indications for and contraindications to endoscopic treatment of bronchial adenoma. There can be no question, however, as to the inadvisability of persisting in the bronchoscopic treatment of adenomas in branch bronchi or of adenomas in any bronchi when there is evidence of extension beyond the wall. Thus, it is clear that lobectomy is indicated for branch bronchus tumors and for most tumors in the upper and middle lobes.

There are two main indications for pneumonectomy for main bronchus tumors: (1) Extension beyond the wall of the bronchus and (2) regional lymph node involvement. According to the evidence which has been presented, the indication is not to be based on the assumption that bronchial adenoma is a precancerous or cancerous lesion. For example, we performed a pneumonectomy on a woman 64 years old for a circumscribed tumor erroneously thought to be a primary cancer of the lung. This proved to be a bronchial adenoma with an adjacent involved lymph node which resembled roentgenologically a circumscribed cancer of the lung. The patient, who was not a good surgical risk, had been virtually symptom-free and had not only carried the lesion for many years, but also would have borne it well indeterminately in all probability. Had we known the nature of the lesion we would have withheld operation despite the involved regional node. To cite instances in which pneumonectomy has not been performed and yet the course has been satisfactory, there are a few patients in whom symptoms subsided

entirely or in large part after partial bronchoscopic removal of the tumor, and in whom death occurred much later from unrelated causes. There are two cases in which drainage of the empyema was the sole procedure and the subsequent course has been satisfactory. Perhaps the advocates of uniform radical operation might, or would, concede conservative treatment in the old or poor-risk patient under certain circumstances. What about the young and good-risk patients who make up the preponderance of the cases? With the present-day development of thoracic surgery virtually no mortality and but little morbidity need be anticipated in such patients whether subjected to lobectomy or pneumonectomy. The argument concerning the malignancy of bronchial adenoma thus becomes, according to this view, merely one of didactic interest. However, it is because most of the patients suffering from bronchial adenoma have many years to live that they should not be deprived unnecessarily of large areas of pulmonary tissue. This discussion may be closed aptly by reference to a patient who has done well after a pneumonectomy for adenoma performed six years ago. The report from another source on the bronchoscopic specimen was "malignant neoplasm." The operative specimen revealed a main bronchus adenoma ideal for endoscopic removal. There is surely no justification for pointing with pride to the result of surgery in this case. To conclude, there is full justification for the conservative treatment of selected cases of adenomas in the main and lesser bronchi, just as the evidence is clear in favor of lobectomy or pneumonectomy in other cases of bronchial adenoma.

SUMMARY

Bronchial adenoma is a clinicopathologic entity. Clinically a syndrome termed the "adenoma syndrome" occurs in the preponderance of cases. The microscopic characteristics are those of a benign neoplasm, with an orderly arrangement of distinctive cells and a virtual absence of mitotic figures. Metastasis is not common and when present it consists usually of limited involvement of an adjacent lymph node, in which the pattern of the parent tumor is repeated. Remote metastases are extremely rare and consist usually of minute deposits which repeat the pattern of the primary tumor. Bronchial adenoma occurs in the larger bronchi in the great majority of cases, rarely in bronchi which are too small to be accessible to the bronchoscope. The tumor may be situated superficially within the bronchus or extend through the wall to a varying degree. Complete eradication of a superficially situated tumor can be achieved by endoscopic treatment particularly when the tumor is situated in a main bronchus. Not only cure but also subsidence of infection and retention of the lung can be achieved thereby. In the case of extension into or beyond the bronchial wall, local excision by bronchotomy as advocated by Eloesser can be considered. The contraindications to bronchoscopic treatment are outlined. The indications for lobectomy or pneumonectomy are stated.

The treatment of bronchial adenoma should be based on the concept of a benign neoplasm with a long life history and not on the concept of a cancerous or precancerous lesion.

DISCUSSION ON "THE MALIGNANT NATURE OF BRONCHIAL ADENOMA" BY DR. ALFRED GOLDMAN,* AND "ADENOMA OF THE BRONCHUS" BY DR. COLEMAN B. RABIN AND DR. HAROLD NEUHOF

DR. EVARTS GRAHAM, St. Louis.—These two papers which we have just heard have been of enormous interest to me, but I must say that they leave me somewhat confused. Many of you will remember that two years ago we heard an extensive discussion of this subject. There were in all 15 or 20 cases reported by various discussors as well as by myself, who gave the paper on the question of so-called adenoma of the bronchus, in which there was undoubted evidence that distant metastases had taken place in these tumors. Now I am very happy to find that Dr. Goldman, I think I am correct in saying, has changed his attitude. Perhaps I am misquoting him, but I believe I am correct in stating that he thought only a few years ago that these tumors were never malignant. I do not know why it is so hard for this idea to die in the minds of everybody, that these tumors should never be considered malignant. Dr. Neuhof has referred to the unwillingness of pathologists to accept the idea that these tumors are potentially malignant, a point which many of you know Dr. Womack and I have been trying to develop successfully for a period of about eleven years now.

Now I do not agree with Dr. Neuhof, if I understood him correctly, that pathologists in general are reluctant to admit the potentiality of malignancy in these cases. I would say, on the contrary, that my own inquiries, not only in the United States but also in England last fall, convinced me that more and more pathologists have come around to the point of view that these tumors are potentially malignant. Perhaps Dr. Neuhof has that idea because he makes the statement, which to me is a very strange one, that he would exclude the cylindromas and the mixed tumors which are malignant and talk only about the adenomas. In other words, he says, "I will dismiss from my talk those which are malignant, and confine my remarks only to those which are not malignant." And yet he mentions that even among the group which he designates as having a benign form of malignancy—whatever that is, I do not know—there is involvement of the lymph nodes, involvement in two instances of the liver, and bronchial wall invasion.

My idea, and my definition of malignancy in a tumor, is evidently totally different from his. It is impossible in a few minutes' time to develop this idea again; I am almost ashamed to stand up here again and redevelop these points which we have emphasized in various articles in the literature over a period of about eleven years. We have never indicated that we thought all these tumors were malignant; we have never indicated that we thought these tumors were malignant at all times. When one considers the question of potential malignancy, one assumes, of course, that something happens to a tumor which is present, the nature of which happening we do not know, but the tumor becomes malignant; something happens to the tumor, sets off a trigger, and it becomes malignant. Now it is entirely possible, of course, that under such circumstances, and there are many other examples in other parts of the body which I will not take time to mention, it is entirely possible that if one sees these tumors in the premalignant stage, of course he will consider them as benign tumors and of course at that time they will be benign tumors. I maintain, however, that the evidence is very strong now that many of these tumors become malignant, and I will state also that I have had three cases of extensive metastases in the liver, of the sort of tumor which Rabin and Neuhof designate as benign adenoma.

I think it is a curious fact, and a very interesting and important one, that except for an occasional and very exceptional report in the literature, nobody knew anything about bronchial adenomas until within the last eighteen or twenty years; in other words, it has been coincidental with development of the bronchoscope. Can one imagine that pathologists, the most famous pathologists in the world in those days, would have failed to see bronchial adenomas; in doing autopsies could one suppose they would have failed to see them? Does

*See page 137 for article by Goldman.

it not seem probable that they would have attracted some attention, because they are interesting tumors, they are curious tumors? Would it perhaps be fair to make a suggestion such as Dr. Womack and I made in our article in 1937, and as Dr. Goldman has also said today, is it not a fair assumption that many of these tumors when they become wildly malignant or definitely malignant make it impossible for the pathologist to recognize that they have originated in an adenoma in the bronchus, because it is impossible to tell after they have spread all over and have involved all the organs. Now, if there were such an occurrence, what sort of tumor would we expect to find? Remember that this adenoma that Dr. Neuhof is talking about is composed of little round cells, scanty cytoplasm, almost entirely nucleus. Dr. McDonald tells us, and shows us a picture, and says that the British call the most malignant type of bronchiogenic carcinoma the oat cell carcinoma. The cell which is seen in these so-called oat cell carcinomas looks exactly like these cells which you see in the bronchial adenomas that Dr. Neuhof is describing. I will pose the question again, then. Is it possible that sometimes these tumors become so malignant that they are recognized only as so-called oat cell carcinomas but which had their origin perhaps in a so-called bronchial adenoma?

One more point which has been mentioned here this afternoon is that these adenomas are characterized by the fact that there is no ulceration in the mucosa overlying. That is true, but even without ulceration of the mucosa overlying I have seen two patients with metastases of the liver from which presumably they died. I have seen other cases with extensive lymph gland involvement. Finally, I would like to make one other statement which is not too conclusive. We have been interested in the very interesting technique which Dr. Harry Green of the Pathology Department at Yale has developed—I am sure many of you are familiar with it—of injecting pieces of tumor into the anterior chamber of the eye of guinea pigs. His contention is that no tissue will grow by such technique except two kinds, malignant tumors and embryonic tissue, and by embryonic tissue he means tissue taken from an embryo. We have recently transplanted so-called adenomatous tissue of the kind Dr. Neuhof is talking about, not the kind he dismisses as being malignant but the kind he says is benign-malignant, into guinea pigs' eyes. Unfortunately, there has not been time for me to find out just what has happened to these guinea pigs, but I had a telegram Saturday afternoon from Dr. Kemler, who has been doing this work, and he said there is a growth in the eyes but he had not yet had time to examine it. I am making that statement for what it is worth. It is not conclusive but we shall report on those findings later after we have had an opportunity to study them.

DR. OSLER ABBOTT, Atlanta.—There is no more intriguing pathology in the lung than so-called adenoma. I also feel that I am not certain what to do about it. I was brought up and trained on the theory that it should be removed by total pneumonectomy. Reports have come out that these tumors may be arrested with lobectomy alone. Our policy in Atlanta had been to do lobectomy for adenoma and if the immediately resected section shows no node involvement, the operation is discontinued at that point. We felt happy with this type of procedure until we encountered a patient a little over a year ago in whom there was an adenoma and in whom considerable lymph gland involvement was noted throughout the mediastinum. A total pneumonectomy was done; there was absolutely no involvement of the glands removed, but a careful examination of the tumor microscopically revealed several microscopic fields in which there was unquestionable transformation of this tumor from its basic cellular structure into a grade 3 squamous cell carcinoma. This experience has been repeated on three occasions since then, and in every case the adenoma has transformed itself into a squamous cell carcinoma with major areas of intact so-called "benign adenoma tissue" about it. Two other patients have been encountered in whom the tumor did not transform itself and there was very extensive invasion of the posterior chest wall on the left side. One patient had extremely extensive invasion of the major mediastinal vessels. I feel that the lesion is only "benign" in its cell structure as it stands, but it is definitely premalignant clinically and can transform into a squamous carcinoma. Whatever type an oat cell carcinoma may be, it could very well have its origin in an adenoma.

DR. CHEVALIER L. JACKSON, Philadelphia.—I remember the occasion Dr. Graham referred to; at that time I reported twenty cases encountered at Temple University Clinic. Since that time we have seen twelve more such patients. Of the original twenty, three had been treated surgically, with lobectomy in two instances and pneumonectomy in one, and seventeen bronchoscopically. At the present time those patients are status quo except for one of those treated bronchoscopically, who has just recently had a very successful lobectomy, with removal of the middle and lower lobes, not because there was progression of the tumor but because she still had recurring attacks of cough with some production due to the bronchiectasis in the lower lobe. Of the twelve new cases, seven patients were treated surgically and five bronchoscopically.

I believe that the crux of the confusion lies in the pathologic and histopathologic diagnosis. I think, as Dr. Neuhof and Dr. Rabin have said, we should eliminate the mixed tumors and cylindromas, and consider these as separate groups. It is extremely interesting to hear Dr. Rabin's view on this, since he is one of the pioneers in the study of these cases. I do not believe true adenomas have any inherent tendency to become malignant, but I do not say they *never* may become malignant. However, I think treatment depends by no means entirely on the pathology; that is, on malignancy or benignancy. Bronchoscopy is always indicated first, for study and biopsy, and for clearing up the lung. Bronchoscopic removal may be curative when the tumor is attached by a narrow pedicle; bronchoscopy will be helpful always when there is need for improved drainage. The indications for pneumonectomy or partial resection are, in my opinion, chiefly in cases where it is impossible to remove the tumor bronchoscopically, and in cases where there has been irreversible damage to the distal portion of the lung as a result of prolonged bronchial obstruction.

DR. ALEXANDER E. W. ADA, New York.—I wish to present two cases which illustrate the problem under discussion. The first is that of a 52-year-old white woman, admitted to St. Luke's Hospital, New York, in May, 1947. She had experienced recurrent hemoptyses for the past twelve years. In December, 1943, bronchoscopy was performed at another hospital and a biopsy was taken from a tumor in the left main bronchus. The microscopic sections were thought to represent adenoma, although certain areas showed definite irregularity in the arrangement of the cells. One year later two successive bronchoscopies were performed in an attempt to remove the tumor. In February, 1947, another bronchoscopic biopsy was reported as adenocarcinoma. Laminographic studies showed a tumor in the lumen of the left lower lobe bronchus, with peribronchial infiltration. Findings on routine x-ray films of the chest were essentially negative. On June 2, 1947, a total pneumonectomy was performed, and recovery was uncomplicated. On gross pathologic examination, a pedunculated rounded tumor, 2.5 by 2 cm., was found just within the opening of the left lower lobe bronchus, projecting into the main branches. The tumor had a depth of 2.5 cm. and extended outward into the surrounding lung. Two centimeters distal to the tumor there was a second one, 2.5 by 1.5 cm. in size, which was similar to the first. The lower lobe showed enlarged thick-walled bronchi and bronchioles with the appearance of bronchiectases and areas of fibrosis.

Sections through the pedunculated portion of the growth showed that toward the surface the tumor was composed of long chains and anastomosing strands of rather small epithelial cells, suggesting an origin from the basal layer of the mucosa. These had a glandular pattern, but the cells were about the same size and shape, stained very deeply and, for thse reasons, did not suggest a very highly malignant growth. In certain areas, however, they had invaded the cartilage of the bronchus and here there was also considerable bone. The latter was surrounded by tumor and the cartilage was detached from the perichondrium. In these areas the lymphatics were invaded, but not the nodes. Pathologic diagnosis was adenocarcinoma of bronchus, nodes not involved.

The clinical course in this patient and the early bronchoscopic and pathologic findings tended to confirm the diagnosis of adenoma. However, over a period of years the tumor seems to have acquired some of the characteristics of malignancy. This case also illustrates the possibility that more than one tumor may be present and escape recognition.

The second patient was a 35-year-old white woman, who was admitted to St. Luke's Hospital in July, 1947. Five years before, she had been hospitalized for a virus pneumonia. Soon after, she developed occasional pain in the left chest, accompanied by cough and hemoptysis. These symptoms persisted one or two weeks at a time and recurred at about monthly intervals, usually at the time of menstruation. Four years before, bronchoscopy had been done, with the diagnosis of adenoma of the lung, although the histology was more typical of lymphoblastoma. Bronchoscopic biopsy one year later was diagnosed carcinoma, and a repeat bronchoscopic biopsy in April, 1947, was diagnosed adenoma.

On admission, the x-ray films of the chest showed no pathologic lesion or abnormality. On bronchoscopy a large papillomatous red mass was visualized, filling the opening of the left upper lobe bronchus. Microscopic diagnosis of a biopsy was adenocarcinoma. On July 15, 1947, a successful left total pneumonectomy was performed.

Pathologic examination showed the tumor to arise from the posterior wall of the upper lobe bronchus. It projected into the lumen and extended toward the hilus and downward into the lingular division. Microscopic examination of the tumor showed it fungating into the lumen, where it was composed of many solid sheets of cells with a glandular arrangement. The tumor cells infiltrated the wall deeply and extended to the cartilage which was partially destroyed. The cells were also outside the cartilage in the peribronchial tissue; however, the nodes were not involved. Pathologic diagnosis was adenocarcinoma of the bronchus; nodes not involved.

These two cases tend to confirm the experience of others, that adenoma of the bronchus may undergo malignant changes, and that certain tumors presenting similar clinical features and bronchoscopic appearance may be be malignant tumors from the onset. It is evident that a correct diagnosis cannot always be obtained from one or more bronchoscopic biopsies. Under the circumstances it would seem that the necessity of pulmonary resection should be of prime consideration and, if performed early in the disease, more conservative excisions or procedures would suffice to effect a cure.

DR. JOSEPH GORDON, Ray Brook, N. Y.—I should like to give a follow-up report on ten cases that have had a period of five years' observation. At the time of the original report by Dr. Chamberlain and myself, seven patients were reported as recovered, two died, and one was inoperable. We now have a report on six cases. Five are considered well, although one has a sinus tract infection. The sixth case is of interest to Dr. Graham. The left lobe of the liver was removed in Albany by Dr. Dickinson, and the tumor cell is identical with that of the bronchial adenoma. This patient has returned to work following lobectomy of the liver.

DR. D. H. WATERMAN, Knoxville, Tenn.—Personally, I agree with Dr. Graham and Dr. Abbott in saying that these are malignant or premalignant tumors. I would like to call attention to the bleeding that occurs on biopsy. As you know, hemoptysis is a very common symptom in the history. We have had four cases in our series in which terrific bleeding occurred when bronchoscopic biopsy was taken, one of these terminating fatally.

I believe Dr. Gordon mentioned that he had had two cases in his series where death occurred at bonchoscopy. We have been rather reluctant to treat or even biopsy these tumors through the bronchoscope when we felt resection should be resorted to anyway.

DR. W. E. ADAMS, Chicago.—A few years ago, Dr. Bloch, Dr. Steiner, and I reported on a group of these patients, and in that group there were three autopsies. One patient died from asphyxia due to obstruction of the trachea by tumor; another from pneumonia; and the third following operation. We called attention to the fact that mediastinal node metastases, and in two of the three, distant metastases to the vertebrae or liver were found. Since that time we have had experience with another patient: a 34-year-old woman who, in 1934, had a circumscribed upper lobe lesion; the sputum was negative. She was given x-ray therapy with complete disappearance of the opacity. She had no symptoms until 1942, nine years later, when hemoptyses began and the lesion appeared as in the original roentgenogram. Re-

section was recommended and was delayed for some months. We found no evidence of involvement of the regional lymph nodes. Eighteen months later she had a pelvic operation, and the liver was found completely studded with nodules. One of these was removed, and showed the same cellular formation as that of the original lesion. She has been completely asymptomatic since operation and shows no signs of metastases. We know, since this has been demonstrated microscopically, that she does have metastases to the liver and we do not know where else.

Experience has led us to believe that these tumors must be considered malignant; slow-growing carcinoma is a good term. From the microscopic appearance one cannot predict which of these lesions will metastasize. Thus, all should be considered potentially malignant. One cannot classify the tumor as to the rate of growth. We know that a squamous cell carcinoma may be present for years without producing symptoms.

Addendum.—This patient recently developed signs and symptoms of cerebral metastases and died shortly thereafter.

DR. EDGAR W. DAVIS, Washington.—Last night and again this morning I talked to several members of this Society about adenomas and to my amazement Dr. Graham a few moments ago used the same expression I heard last night, "We are confused." To listen to the various discussions on the management of adenomas is often more confusing. It would seem that it is virtually impossible to obtain uniform opinions from various pathologists as to the malignancy or benignancy of these lesions. It is nice to have a correct preoperative diagnosis on a patient but I do not agree that this is a clinicopathologic entity because I find from bronchoscopic biopsy and even from the surgically removed specimen that pathologists of repute differ widely in defining the nature of the lesion. It is for this reason that I doubt that the histologic report of a biopsy should necessarily dictate our mode of therapy. One pathologist may classify a given section as a grade 1 or grade 2 malignancy, whereas another pathologist may classify it as a benign lesion.

It has been our policy in the management of these so-called adenomas to take a bronchoscopic biopsy if possible. If all the tumor can be removed bronchoscopically and if all the sections by different pathologists are called benign, nothing further is done. If, however, there is any question as to whether it is a benign lesion or if it is not entirely accessible to the bronchoscope, a lobectomy or segmental resection is done. If the pathologist then reports a benign lesion it is unnecessary to sacrifice any additional lung tissue. If it is reported as questionably malignant, we do not believe anything short of a lobectomy should be done.

DR. CLARENCE CRAFOORD, Stockholm. —We have one case in which operation was done in 1936 with total pneumonectomy, which in some respects illustrates the question of malignancy of bronchial adenomas. At the time of operation the diagnosis was bronchial carcinoma. There was invasive growth in the mediastinum and metastases were found in mediastinal lymph nodes. A study later on of the specimen, however, showed that this tumor without doubt belonged to the group of mixed salivary gland tumors to which group we consider the so-called bronchial adenoma to belong. That patient was well until 1946, when he came back with recurrence in the bronchial stump and with clinical evidence of tumor growth in the mediastinum. He died about one year later. In this case there were undoubtedly clinical signs of the bronchial tumor so far back as seven years before admission. This case and many more in our experience have shown us that if tumors of this type are to be considered as potentially malignant, they cannot be clinically regarded malignant in the same sense as real carcinomas. If within a reasonable time they can be locally radically excised, the risk of a recurrence must be considered as very small. Because of that we have now come to the conclusion that it is advisable to treat these tumors more conservatively than carcinomas. As it is impossible to diagnose if there is in addition to the intrabronchial bronchoscopically visible growth also an extrabronchial extension in the great majority of cases, we are now of the opinion that endobronchial removal of the tumor is not to be con-

sidered as a sane procedure, with the possible exception of very rare cases. Therefore, we now advise an exploratory thoracotomy. If the tumor has not produced irreversible changes in the lung peripheral to where it is localized in the bronchial tree we try to remove radically but locally both the intra- and extrabronchial part of the tumor with its surrounding "capsule" of connective tissue and the piece of bronchial wall that is invaded by the tumor. The incision in the bronchus is planned in such a way as to give the best opportunity for a plastic reconstruction of the bronchus without excising any part of the lung.

We recently saw a tumor of this kind in a young man. The tumor was extensively infiltrating a part of the bronchial wall from just above the upper lobe bronchus down to below the middle lobe bronchus. When he entered the hospital he had a total atelectasis of the lung and we thought that a total pneumonectomy was going to be necessary. By bronchoscopic removal of a part of the tumor the lung became normally aerated and on subsequent x-ray views the chest seemed to be normal. At thoracotomy it was possible to do a radical local excision of the extrabronchial plum-sized part of the tumor, the infiltrated part of the bronchial wall, and the remains of the intrabronchial part of the tumor with subsequent plastic repair of the bronchus. The immediate postoperative course was without complications and the patient healed with a normally functioning lung instead of having a pneumonectomy done. There has been a tendency of stricture formation at the site of the bronchial repair which has made bronchoscopic treatment at intervals with dilatation necessary in the beginning of the postoperative period. The tendency to stricture formation has gradually diminished. I know of one more such case. This was an officer attached to the Royal Air Force in England. He was going to be excluded from further active service if a total pneumonectomy was done. He was operated upon by Mr. Price Thomas in the same manner as just described. This was about two years ago and he is still well, with a functioning lung and in active air service. I think that local excision of the bronchial wall with the tumor is the sound procedure in such cases.

DR. JEROME R. HEAD, Chicago.—The question under discussion is very important. The answer to it will determine whether individuals with these tumors are to be subjected to radical surgery, often to pneumonectomy, or to bronchoscopic removal of the growth, or to nothing. The matter is definitely confused. We insist on talking as if there were only one type of adenoma, when in reality ther are two types; and we are confused concerning the definition of malignancy. The present papers have been particularly enlightening in that they have made a sharp distinction between cylindromas, which are potentially malignant, and adenomas, which are essentially benign. Dr. Graham uses the world "malignant" from the point of view of the microscopist and the gross pathologist. He says that these tumors are malignant because some of the cells are undergoing mitosis, because they invade lung and bronchus, and because metastases are sometimes found in the hilus lymph glands. The malignancy of a tumor can be judged only by its effect upon the patient. If the rate of cell division and tumor growth and invasion and metastasis are so slow that the patient will not be injured during his lifetime, then the tumor is clinically benign however malignant it may look or be according to any other standard of malignancy.

In Chicago recently Dr. Holinger presented a paper reviewing his cases of bronchial adenoma and in the discussion many more cases were reported upon. In this entire group of approximately 60 cases, many of them with histories extending over twenty or thirty years, the only patients who had died had done so as a result of surgery. (Since that time one patient with cylindroma has died of metastases to the liver.)

In the present discussion two cases have been reported in which adenomas, of known long standing, are said to have become malignant. The only evidence for this was microscopic and the only authority was the word of the particular pathologist. We all know that microscopically adenoma resembles carcinoma very closely. Very frequently a pathologist mistakenly calls an adenoma a carcinoma. To say, then, that because at one time one pathologist called a particular tumor an adenoma and that at another later time another pathologist

called it a carcinoma—on this evidence to say that the tumor has changed and become malignant is not justified.

Dr. Graham suggests that it is possible and even probable that many of the oat cell and squamous cell carcinomas which we encounter have developed from formerly benign adenomas. This speculation does not accord with clinical facts. If it were true, we would encounter many cases of carcinoma with a history of hemoptysis and recurring atelectasis going back over many years. We do not encounter such cases; in fact, if a patient suspected of having cancer states that he coughed up blood two or four years before, we take this as fairly conclusive evidence that the present trouble is not cancer.

DR. CARL EGGERS, New York.—If we consider adenoma of the bronchus a really malignant tumor, it follows that a radical procedure is indicated. The question comes up, what is one's personal experience with this growth and what is the experience of the profession in general. I have made a few observations which have shown that the prognosis does not alone depend on the degree of malignancy of the growth but on other factors, such as hemorrhage or infection.

Some years ago I had under my care a patient with extensive unilateral bronchiectasis of unknown etiology. She had been treated with cauterization and gradual destruction of the lung. One day she had a severe hemorrhage and died. At autopsy it was found that she had a large tumor of the bronchus which could not have been removed because of extension beyond the bronchi into the pulmonary tissue. A diagnosis had not been made because the bronchoscope was not known at that time.

Some time later I had a boy under my care who had extensive bronchiectasis with recurrent attacks of high fever. Operation was refused for a long time. Eventually he was operated upon because of continued sepsis. He died of a cerebral embolus. Autopsy showed a bronchial adenoma which could not have been removed locally.

Both these patients died not as the result of the high degree of malignancy of the growth, but of complications caused by the presence of the tumor.

About twelve years ago Dr. John Kernan of New York collected a number of cases of adenoma of the bronchus. He watched and treated these patients for sometime, with the object of causing destruction of the growth and maintaining a free airway. The first patient he carried along in that manner for about twelve years with the aid of electrocoagulation and later radon seeds implantation. Eventually the growth could no longer be controlled and stenosis of the bronchus was threatened. The biopsy examination showed carcinoma. A pneumonectomy was done, resulting in cure. The growth had extended beyond the cartilaginous rings into the pulmonary tissue.

From the pathologists' standpoint these tumors are malignant because they extend beyond the bronchial wall into the pulmonary tissue. However, they do not break through the basement membrane, but push the tissue ahead of them. Although called malignant, they are of a low grade of malignancy.

It is my feeling that the treatment of bronchial adenoma, whether called truly malignant or not, requires good judgment. The important thing is to maintain a good open airway in order to avoid the danger of bronchiectasis. If that can be done by the removal of a small, somewhat pedunculated growth, conservatism may be justified. If after local removal the growth recurs and there is danger of retention and infection, a more radical procedure becomes indicated.

DR. E. J. O'BRIEN, Detroit.—I hesitate to get into this prolonged argument, but since it has turned into a psychiatric discussion I think I am well qualified. I have had in mind all afternoon one case which was reported by Dr. Holinger and Dr. Clerf as an adenoma of the bronchus removed through a bronchoscope. I saw this patient a number of years later in the operating room of Dr. Churchill in Boston. The whole lung was so involved then that a pneumonectomy was necessary. This sounds malignant to me.

DR. GOLDMAN.—There is not time to answer all the problems raised. I have changed my mind from what I thought at the time I read the paper before the Society in 1938. We had never observed up to that time any lymph node metastases, and we followed some patients for twenty years. The first patient reported in that paper was operated upon about 1915, when empyema and abscess were drained. The tumor was as large as a grapefruit when the patient died. There was no lymphoid involvement.

I have modified my opinion to this extent. There are varieties of adenoma. One variety which was shown is cylindroma or mixed tumor. Of this variety shown in the first case, I have seen two cases. The other patient also died after bronchoscopic removal of the tumor. Early surgery with removal was not done. I believe we should differentiate the adenoma group from cancer, if for no other reason than that when we operate and remove the tumor the patient's chances are so much better, and I believe we should always separate them from carcinoma in reporting statistics.

I am somewhat pleased to find the evidence as reported by Dr. Abbott and Dr. Gordon, that these tumors may metastasize and go for many years without producing death. It seems to me they metastasize whether they are old or young. It seems local lymph node metastasis does not always occur, but is perhaps more often found in older tumors. Regarding what Dr. Crafoord said, I reported a case in 1947 which had primary bronchoscopic removal, and after three years there was local bronchial recurrence. When a lower left lobe resection was done a tumor nodule could be seen inside and outside the bronchus. This patient has had symptoms for twenty-five years and there is no sign of metastases. If in a given case all the tumor can be removed by bronchotomy, local surgical resection, there are patients in whom that can be done to conserve the lung. Regarding Dr. Eggers' cases I do not agree completely that local removal is always successful. I have seen deaths from attempt at bronchoscopic removal. I am sure every bronchoscopist is alarmed by the large amount of bleeding which may occur. Particularly where there is suppuration, opening the bronchus will not cure that patient any more than a chronic abscess, and resection should still be done for the original disease.

There is much more to be said about his subject. The ideas of the origin, and the ideas we have as to the growth of these tumors may be modified. Dr. Nathan Friedman has an idea about mixed tumors. He considers that these tumors, arising from the original cell, may form two or more different cells—and they do not have to go through a complete life cycle to complete malignancy—they may die out; thus some adenomas may develop to a certain point and go no further, and perhaps others go to complete malignancy.

DR. RABIN.—In this prolonged discussion it has been emphasized that certain members have been left with a feeling of confusion. I may say this: where confusion begins, this is the beginning of clarification. Years ago this tumor was reported simply as carcinoma and therefore did not constitute a special problem. Dr. Harry Wessler, to whom belongs the credit for recognition of the tumor, was confused when the pathologist reported several such tumors of long standing in young women as malignant. Dr. Mandelbaum admitted the growths were peculiar and different from carcinoma, and he said: "If it is shown to me that they run a benign course, we must consider them benign, even though to me as a pathologist they appear malignant."

We have passed a certain phase in the confusion regarding these growths, that is, confusion with ordinary forms of carcinoma. We have not passed the point of confusion of the adenoma with other unusual tumors. A few slides will illustrate this: (Slides.) The type shown on the screen falls into the group called the carcinoid type of adenoma which, together with the acinar type, make up the category of bronchial adenoma. I would exclude the cylindroma, which looks like certain portions of salivary gland tumors, mixed tumors containing bone and cartilage and sometimes other elements, and the entire group of mixed tumors, including osteochondroma. In addition, tumors resembling certain basal cell carcinomas of the skin showing cystic adenomatous changes are more properly relegated to carcinoma or cylindroma and should also be excluded.

Before formulating a clinical classification of the growth one must start with a pathologic classification. The pathologist must classify what he sees microscopically, and then follow the pathologic course of the growth before he can help the clinician in planning treatment.

In this respect I wish to show the following slides. This table shows five cases with tumors in the main bronchus, and no response to endoscopic therapy. They show a history of from seven to thirty-seven years, and how much longer the tumors were present we do not know. There we have failure of endoscopic removal in patients who have a long history averaging thirty-three years of symptoms. Here we have a group of fifteen patients who appear to be well after bronchoscopic treatment and who have been followd for a considerable time, without recurrence. One has been followed for fourteen years, and five have been followed for more than ten years without recurrence being found. It must be concluded that at least some of these patients are cured by simple endoscopic removal.

We are dealing with a growth different from carcinoma. We know the history is longer; we know the sex incidence is different from carcinoma. It comprises a distinct pathologic entity. Whether this tumor is amenable to a minor form of treatment or whether it requires resection of a large amount of tissue is not basic. We must separate from the adenoma other tumors that have a definite pathologic picture relating to other growths. When we do that we find the incidence of what has been called malignant change diminishes.

I do not think we have to define malignancy. We have to know what the growth does. Here is a tumor that grows for a long time (we have one-half dozen patients who have had the tumor for thirty years)—a tumor which remains separated from the remainder of the bronchial wall in most cases, but in some cases does grow through the wall and even into the lung, occasionally involving the adjacent lymph nodes, and sometimes causes distant metastases, the tumor tissue still maintaining a benign appearance. These are not the characteristics of carcinoma in general and calling it carcinoma makes for confusion. We do not know what term is best to attach to this growth. The word adenoma is not a good one but should be retained until a better one is found.

There is little basis for considering that many carcinomas have their origin in bronchial adenomas. Dr. Head has pointed out that the duration of life in bronchial carcinoma is uniformly short. The only type of carcinoma that has a similarity to bronchial adenoma is the small cell carcinoma. These have the shortest history and this together with the absence of a polypoid appearance to these growths excludes the possibility of their origin in a bronchial adenoma. The paucity of reports of autopsied cases of adenomas must have some other explanation than that the adenomas have changed to carcinomas by the time the patients die.

THE CLINICAL USE OF A PROSTHESIS TO PREVENT OVERDISTENTION OF THE REMAINING LUNG FOLLOWING PNEUMONECTOMY

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AND J. A. COCKE, M.D.
PHILADELPHIA, PA.

WHEN the chest is closed without drainage following a pneumonectomy, the pleural cavity gradually becomes filled with blood and serum. In time, this fluid may be absorbed or it clots and contracts and, as a result, the mediastinum is drawn over to the affected side. This may produce considerable overdistention of the remaining lung (Figs. 1 and 2). This phenomenon may be prevented by a thoracoplasty done electively, or to obliterate the empyema cavity in case of infection (Fig. 3).

It has now been amply demonstrated that most patients do fairly well following a pneumonectomy without a thoracoplasty in spite of emphysema of the remaining lung. Many thoracic surgeons have had the impression that patients subjected to a thoracoplasty have had a better clinical result than those who were allowed to develop overdistention of the remaining lung. Cournand and his associates[1, 2] have presented data which tend to confirm this impression. They found both in children and in adults that the exercise tolerance was better in those patients in whom overdistention of the remaining lung was prevented by thoracoplasty.

Even though the thoracic surgeon agrees that overdistention of the remaining lung following pneumonectomy is undesirable, he may hesitate to subject his patient to an additional procedure as major as a thoracoplasty. If a simpler method of preventing mediastinal shift were available, it might be more widely used.

In a preliminary report,[3] we pointed out that overdistention of the remaining lung following pneumonectomy in dogs could be prevented by filling the pleural cavity with hollow plastic balls made of methyl methacrylate (Lucite).* In the dogs sacrificed after four months, there seemed to be so little foreign body reaction to the Lucite balls that a clinical trial appeared to be warranted. Our present report concerns observations made in dogs sacrificed one year after placing Lucite balls in the pleural cavity following pneumonectomy, as well as clinical experience with the use of this prosthesis in eight patients following pneumonectomy for carcinoma.

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Read at the Twenty-eighth Annual Meeting of The American Association for Thoracic Surgery, Quebec, Canada, May 31, June 1, 2, 1948.

*The Lucite balls used in these experiments were obtained from the Nichol Product Company, Moorestown, N. J.

Fig. 1.

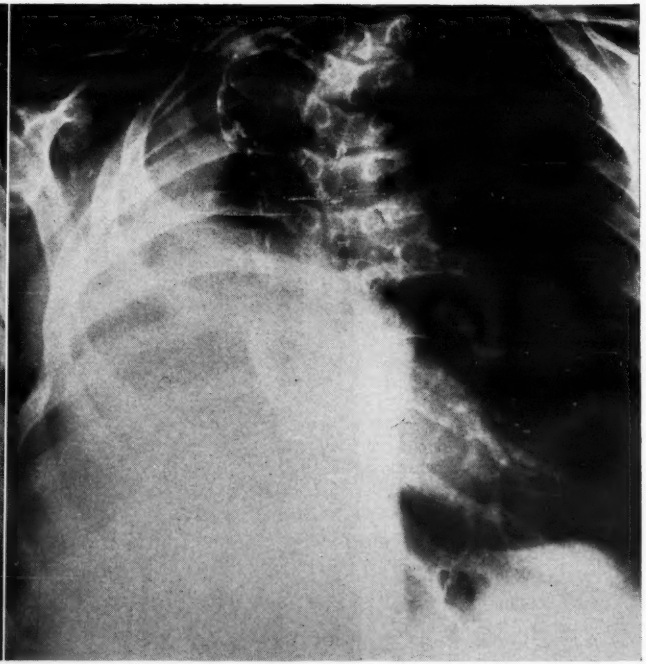


Fig. 2.

Fig. 1.—Severe overdistention of the right lung following left pneumonectomy for bronchial adenoma in a 50-year-old man. Barium in the esophagus shows it to be pulled over to the left. The right lung approaches the left side of the chest wall. (From Johnson and others: Surgery, August, 1947.[3])

Fig. 2.—Severe overdistention of the left lung following right pneumonectomy for carcinoma in a 58-year-old man. This man has had a five-year cure and complains only of some dyspnea on exertion. (From Johnson and others: Surgery, August, 1947.[3])

ANIMAL EXPERIMENTATION

As mentioned in our previous report, when no prosthesis was placed in the pleural cavity, the remaining lung became overdistended and partly filled the pleural cavity on the opposite side (Fig. 4). When Lucite balls were placed in the pleural cavity after removal of the lung, the mediastinum was maintained in the midline and overdistention of the remaining lung did not occur (Fig. 5, *A* and *B*). Since the previous report, four dogs were sacrificed one year following the insertion of the prosthesis. Four dogs remaining are living and apparently well approximately eighteen months after operation.

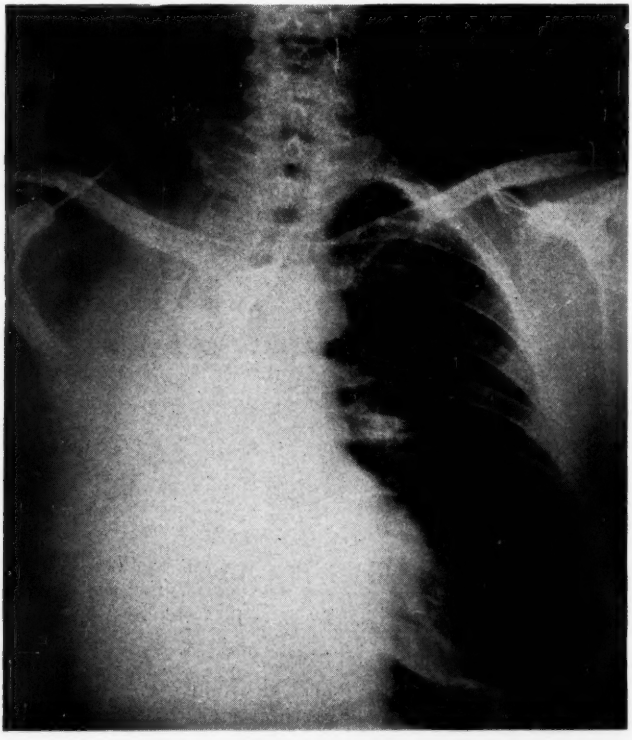


Fig. 3.—One year following right pneumonectomy and thoracoplasty for carcinoma of the lung in a 54-year-old man. The mediastinum is maintained near the midline and severe overdistention of the remaining lung is avoided. (From Johnson and others: Surgery, August, 1947.[3])

In all of the dogs the Lucite balls could be heard with a stethoscope to rattle with each heart beat. At autopsy in one dog each ball was found to be almost completely encapsulated by fibrous tissue with no fluid between the balls (Fig. 6). As seen in Fig. 7, the balls were still in contact at some points. Microscopic examination showed the tissue covering the balls to be acellular fibrous tissue with very little foreign body reaction (Fig. 8).

In the other three dogs, the Lucite balls were encapsulated as a group (Fig. 9). It appeared that each ball had its own niche as evidenced by the

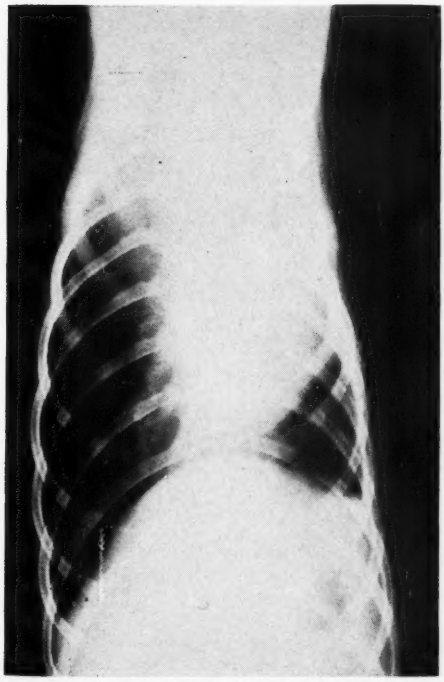
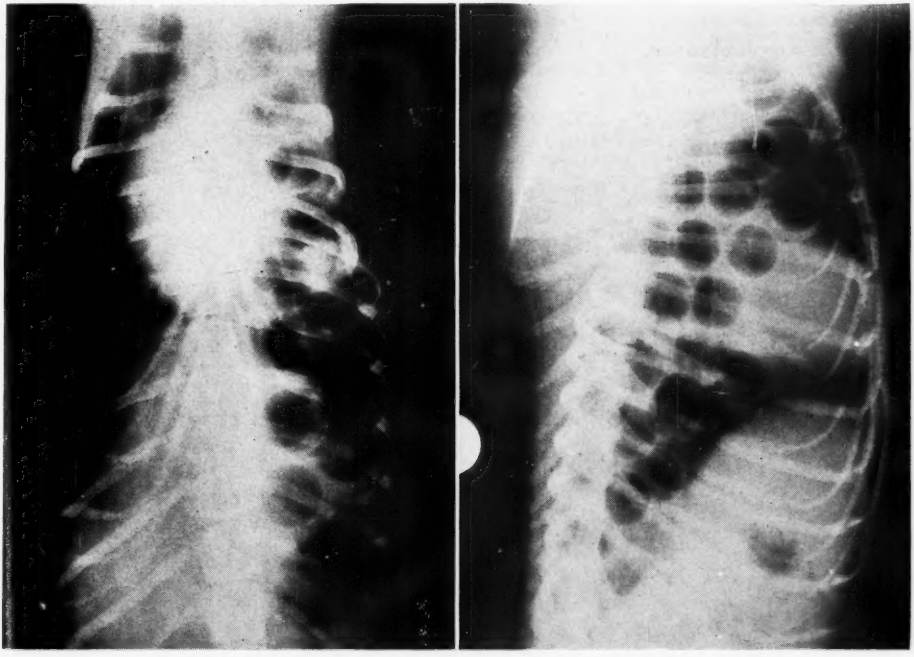


Fig. 4.—A dog two months following left pneumonectomy without the use of a prosthesis. The mediastinum is over against the left side of the chest wall and the overdistended right lung fills the lower part of the left side of the chest cavity. (From Johnson and others: Surgery, August, 1947.[3])



A. *B.*

Fig. 5, *A* and *B*.—Anteroposterior and lateral views of a dog after left pneumonectomy with twenty-four Lucite balls in the pleural cavity as a prosthesis. (From Johnson and others: Surgery, August, 1947.[3])

Fig. 6. Fig. 7.

Fig. 6.—Dog sacrificed one year after pneumonectomy and insertion of Lucite ball prosthesis. In this dog the balls were almost encapsulated individually with fibrous tissue. There was no free fluid present.

Fig. 7.—Same as Fig. 6, showing that there was still contact between the balls at some points.

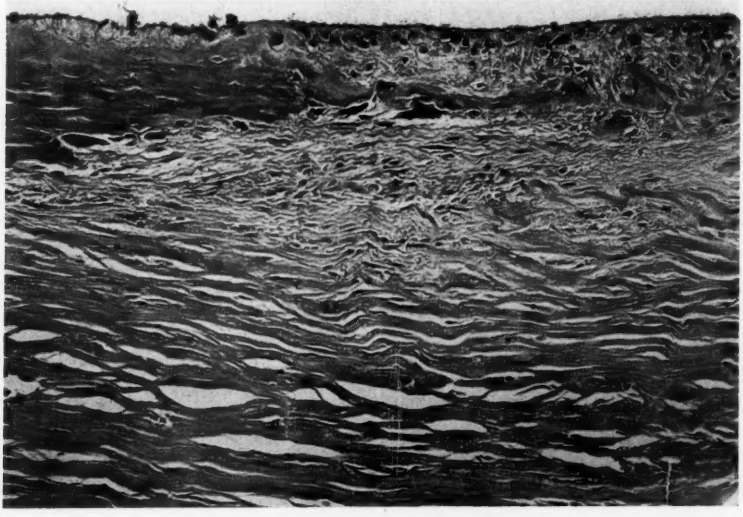


Fig. 8.—Photomicrograph of avascular fibrous tissue encapsulating Lucite balls in dog shown in Figs. 6 and 7.

Fig. 9.—Dog sacrificed one year after pneumonectomy and insertion of Lucite ball prosthesis. In this dog the balls were encapsulated as a group. The space between the balls was occupied by clear fluid.

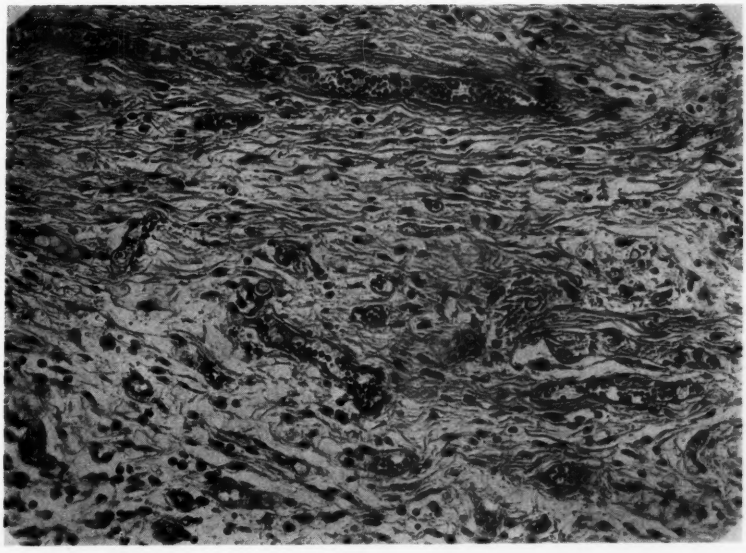
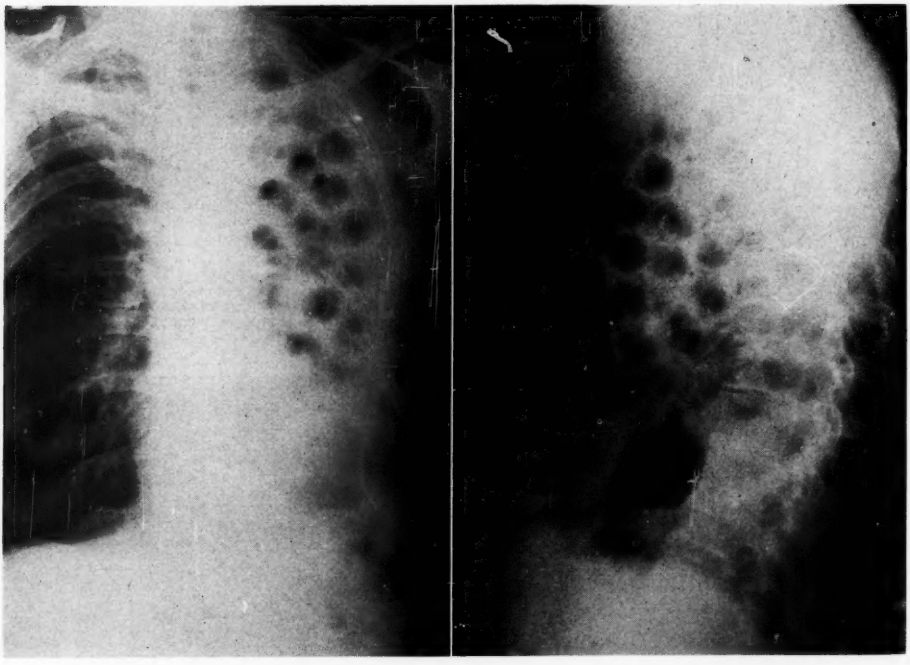


Fig. 10.—Photomicrograph of tissue removed from the pleura between the points in contact with the balls in dog shown in Fig. 9. This tissue is very vascular with cellular infiltration.

impressions on the surrounding pleura. The space between the balls was filled with clear fluid. Microscopic examination of the smooth pleura where a ball was in contact with it was similar to that in Fig. 8. On the other hand, the pleural surface between the balls had the appearance of granulation tissue (Fig. 10). The capsule surrounding the entire group of balls was thick and avascular.

A Lucite ball prosthesis seemed to be the most satisfactory of the prostheses considered for the following reasons: (1) conformity to a pleural cavity of any size or shape by varying the number of balls; (2) lack of evidence of increased infection in the pleural cavity; (3) minimal foreign body reaction; (4) ease of removal through a small incision in the event of infection; and (5) lightness of weight.



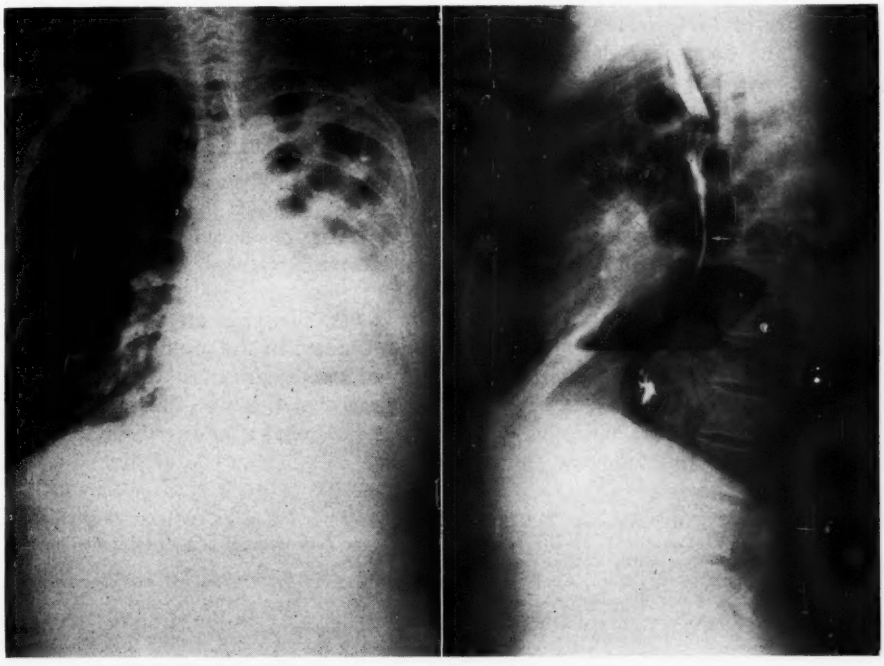
A. *B.*

Fig. 11, *A* and *B*.—Anteroposterior and lateral chest films of a patient with a Lucite ball prosthesis eight months after pneumonectomy. Seventy-five balls were used. There is no overdistention of the remaining lung.

The patients chosen for clinical trial had pneumonectomies for carcinoma. It seemed advisable to try the prosthesis cautiously at first in patients whose life expectancy was short in order to exclude possible unforeseen complications before using it in patients with a longer life expectancy.

The Lucite ball prosthesis has been placed in the pleural cavities of eight patients following pneumonectomy. Inasmuch as when a thoracoplasty is done to prevent mediastinal shift, portions of only seven or eight ribs are usually

resected, the pleural cavity has not been completely filled with balls in these patients. The number of Lucite balls used has varied from thirty-five to seventy-five. The patient with seventy-five balls in the left side of the chest was operated upon about eight months ago. Recent x-ray pictures show the mediastinum to be in the midline and no overdistention of the remaining lung (Fig. 11, *A* and *B*). In a smaller individual, thirty-five balls did not fill the pleural cavity but have prevented overdistention of the remaining lung (Fig. 12, *A* and *B*).



A. *B.*

Fig. 12, *A* and *B*.—Anteroposterior and lateral chest film of a patient with a Lucite ball prosthesis six months after pneumonectomy. Only thirty-five one-inch balls were used. The mediastinum is maintained in the midline.

Comment.—Immediately after operation, when the Lucite balls were surrounded by air, one could occasionally hear them rattle a foot or two away from the patient. At that stage, the patient as well often heard the balls rattle. When the air was replaced with fluid, the patients were no longer conscious of the noise. With the stethoscope, however, the rattle may still be heard with each beat of the heart in all of the patients.

There has been no evidence that the balls have caused increased postoperative pain. In two of three patients who complained of more pain than usual, we were able to demonstrate metastatic lesions. The possibility of increased pleural effusion due to the presence of the balls was considered. Thoracenteses were done whenever required to maintain the mediastinum in the midline or slightly toward the operated side. In three of the patients, thoracentesis was

never required. Two of the patients were tapped only once or twice. Three of the patients required thoracentesis as late as seven to ten days after operation but it could not be determined whether the presence of the prosthesis played a part in this.

In none of the eight patients has there been any postoperative infection or unusual febrile response. In all of the patients the prosthesis has accomplished the objective of maintaining the mediastinum near the midline. Although we have been looking for possible hazards or complications resulting from the presence of the prosthesis in the pleural cavity, none has yet been apparent. Since the dogs have been observed for only eighteen months and the first patient for only eight months, late complications of foreign body reaction may yet develop.* Our experience to date suggests that the Lucite balls may prove to be an effective and safe substitute for elective postpneumonectomy thoracoplasty.

CONCLUSIONS

1. Lucite balls placed in the pleural cavity of dogs following pneumonectomy prevented mediastinal shift and were well tolerated as evidenced by autopsy studies one year after operation.

2. In eight patients in whom Lucite balls were placed in the pleural cavity after pneumonectomy the mediastinum has remained in the midline and there have been no apparent complications resulting from the presence of the balls.

3. The evidence presented to date suggests that the use of a Lucite ball prosthesis may be a simple and effective method of preventing overdistention of the remaining lung following pneumonectomy.

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(See discussion on page 187.)

*It has now been nineteen months since the first patient was operated upon and no complications have developed. In some of the patients the balls no longer rattle.

AN EVALUATION OF EXTRAPLEURAL PNEUMONOLYSIS WITH LUCITE PLOMBAGE

Report of Fifty-one Cases

Josiah C. Trent, M.D.,† and James D. Moody, M.D. (by invitation), Durham, N. C., and Joseph S. Hiatt, Jr., M.D. (by invitation), McCain, N. C.

SINCE Tuffier first performed his "pleuroparietal stripping" in 1891, the operation with modifications has been used by thoracic surgeons all over the world. To maintain the collapse effected by extrapleural separation of the parietal pleura from the chest wall, a multitude of substances has been used including air, fat, muscle, paraffin, oil, gauze, cellophane, rubber, fiber glass wool, and many others. The search for the ideal substance goes on.

In 1945 Wilson and Baker[1] found that Lucite (methyl methacrylate) in the form of spheres was inert in the extrapleural space in the experimental animal and maintained satisfactory collapse of the lung. The results of this study were sufficiently encouraging to justify clinical trial.

The first patients selected for operation were "those so-called good chronics in whom thoracoplasty was contraindicated because of low vital capacity, advanced age, bilateral cavities or those who had an unsuccessful thoracoplasty." The simplicity of the operation and the immediate good results appealed to patient and surgeon alike, and many patients refused to have any but the "ball" operation. Also, as experience with the procedure was gained, the indications for operation were broadened to include those patients in whom a thoracoplasty would have been the operation of choice.

A preliminary report by Wilson[2] of the first fifteen patients on whom follow-up study was carried out for six to ten months showed thirteen improved, one unimproved, and one dead. In spite of the early good results, however, Wilson emphasized the experimental nature of the operation and warned against excessive optimism. From January, 1946, to November, 1946, thirty-six additional patients were operated upon by members of the Duke Hospital staff. As late complications began to occur the operation was discontinued in January, 1947, until adequate follow-up studies could be made and the procedure evaluated. This report is based on an analysis of fifty-one cases of extrapleural pneumonolysis with Lucite plombage in white patients operated upon one year and six months to two years and ten months prior to this report. We feel that it is highly important to report our experiences with the operation at this time, for the practice of inserting Lucite spheres in the chest is spreading and may go far before attention is called to its dangers.

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Read at the Twenty-eighth Annual Meeting of The American Association for Thoracic Surgery, Quebec, Canada, May 31, June 1, 2, 1948.

†Dr. Trent died on Dec. 10, 1948.

In Table I are presented the number of operative cases in each age group, their classification, and the type of operation which after a careful review of the clinical history and roentgenograms we feel was indicated in each case. Nearly all of the cases fall within the far-advanced class. All patients except three had bilateral involvement. Twelve patients had had a previous thoracoplasty. Nineteen had bilateral cavities or were suspected of having bilateral cavities. Thirteen patients were considered unsuitable for any type of surgical collapse because of age, extent of the disease, and clinical condition. In only six, in retrospect, would we have considered extrapleural pneumonolysis with plombage.

TABLE I. FIFTY-ONE CASES, THIRTY-FIVE MEN, SIXTEEN WOMEN

AGE (YR.)	NO. OF CASES	CLASSIFICATION			TYPE OF OPERATION INDICATED			
		M.A.*	F.A.†	THORACOPLASTY	THORACOPLASTY	REVISION	PLOMBAGE	NONE
20-30	10	3	7	1	7		1	2
30-40	13	1	12	5	6	2	2	3
40-50	16	2	14	5	8	3	2	3
50-60	9		9	1	3	1	1	4
60-70	3		3		2			1
Total	51	6 (11.8%)	45 (88.2%)	12 (23.5%)	26 (50.9%)	6 (11.8%)	6 (11.8%)	13 (25.5%)

*M.A., Moderately advanced.
†F.A., Far advanced.

The technique of the operation has been described in detail previously by Wilson.[2] In the present series the anterior approach was used forty-four times and the posterior approach seventeen times. Three of the patients had a two-stage unilateral procedure while four had a bilateral procedure.

COMPLICATIONS

Complications of the operation may be divided into two groups, those occurring during operation or in the immediate postoperative period, and those occurring several months to years after the initial procedure.

A summary of the early complications may be found in Table II. Thirteen patients had moderate to severe dyspnea which gradually subsided in all except four. In four patients the cavity was entered at the operating table. Only one of these patients survived. In three cases a mediastinal shift was observed. In one of these the spheres were inserted under a thoracoplasty and produced

TABLE II. OPERATIVE AND EARLY POSTOPERATIVE COMPLICATIONS (TWENTY CASES, 39.3 PER CENT)

COMPLICATION	NUMBER	PER CENT
Dyspnea (moderate to severe)	13	25.5
Cavity entered	4	7.8
Mediastinal shift	3	5.9
Spread	2	
Dissection downward of spheres	2	
Compression of superior vena cava	1	
Cardiac failure	1	
Urticaria	1	
Wound separation	1	

only a moderate shift. In the other two which terminated fatally the balls dissected caudally and into the mediastinum. In two patients a postoperative spread of the disease occurred. One patient died of cardiac failure, probably as a result of reduction of the pulmonary vascular bed. In one patient, immediately after operation marked engorgement of the neck and arm veins was noted. He was returned to the operating room and after removal of one sphere the engorgement was relieved. Trial reinsertion of the sphere produced the same engorgement. This phenomenon was thought to be due to compression of the superior vena cava. One patient on the tenth postoperative day developed a generalized urticaria associated with an eosinophilia which subsided spontaneously. In one patient the wound separated before the patient's discharge from the hospital. This patient later developed a bronchocutaneous fistula.

The late complications are summarized in Table III. The tuberculous infections of the extrapleural space were diagnosed by aspiration, the presence of a sinus, or reoperation. Four patients remained dyspneic after operation. Two patients had severe incapacitating chest pain. One of these became a narcotic addict before death; the other developed a tuberculous infection around the spheres two years after operation. In one patient roentgenograms showed the spheres protruding through the mediastinum into the opposite side.

Because of the difficulty in visualizing a cavity in the lung once the Lucite spheres have been inserted, the number of patients with persistent cavities under the spheres cannot be determined accurately even with the aid of laminography. A fluid level in one or more of the spheres is a frequent roentgen finding and may be a source of confusion in attempting to identify cavities. In three pa-

TABLE III. LATE POSTOPERATIVE COMPLICATIONS (SIXTEEN CASES, 31.3 PER CENT)

COMPLICATIONS	NUMBER	PER CENT
Tuberculous infection around spheres	11	21.5
Draining sinus	4	7.8
Spheres extruded beneath skin	4	7.8
Dyspnea	4	7.8
Severe chest pain at operative site	2	
Migration of spheres into mediastinum	1	

tients in whom definite cavities could be seen under the spheres, an attempt was made to remove the spheres preliminary to further surgical collapse or resection. In one case the spheres were so densely embedded that removal was impossible without endangering the patient's life. The attempt was abandoned after five spheres had been removed and the cavity had been entered. In the other two cases the spheres were removed with great difficulty. In each case tuberculous granulation tissue was present between the spheres. One of these patients had a fatal pulmonary hemorrhage a few days after operation, while the other developed a severe tuberculous wound infection. In five patients the spheres were removed because of tuberculous infection in the extrapleural space which developed two months to two years after operation. In most of these cases removal was relatively easy, but in two cases all of the spheres were not recovered. One of these patients was subjected to a second operation to recover a sphere

TABLE IV. RESULTS IN FIFTY-ONE CASES

AGE (YR.)	IMPROVED	UNIMPROVED	WORSE	DEAD	
				OPERATIVE	LATE
20-30	2	1	2	1	3
30-40	3	1	6	1	3
40-50	3	1	8	3	1
50-60	1	0	2	3	3
60-70	1	0	2	0	0
	10 (19.6%)	3 (5.8%)	20 (39.2%)	8 (15.7%)	10 (19.6%)

Total mortality 35.3%

embedded in the apex of the lung which could not be found at the first operation. Four of these patients had draining sinuses and one an open wound. One patient who developed a tuberculous infection of the extrapleural space two months after operation had a wound separation and a fatal hemorrhage from the wound.

RESULTS

We have considered as improved those patients who improved symptomatically and who had negative sputum; as unimproved (two patients) those who were not made worse by the operation but who continued to have symptoms and unsatisfactory sputum examinations; as worse those with positive sputum and continued progression of the disease. The complete results are summarized in Table IV. Ten cases can be classified as improved. These will be given in detail. Twenty were definitely worse and seventeen patients died as a direct result of the operation; one patient died two years after operation from coronary occlusion (no autopsy). The corrected late results in forty-two cases are summarized in Table V. Excluded from this table are eight operative deaths and one recovery in which no spheres were inserted following entry of the cavity.

Eight cases in which death occurred from seven to sixty days postoperatively are considered operative deaths. All these patients were poor operative risks, four of them hopelessly ill. In three of these cases the cavity was entered and no spheres were inserted. Death resulted from infection and respiratory failure. In three cases the spheres were removed six to eight days after operation because of marked dyspnea. One patient died as the spheres were being removed on the tenth day. Another patient died of circulatory failure attributed to a reduction in size of the pulmonary vascular bed.

Of the ten late deaths which occurred from two months to two years postoperatively, four resulted from respiratory failure, one from hemorrhage from

TABLE V. CORRECTED RESULTS IN FORTY-TWO CASES*

AGE (YR.)	IMPROVED	UNIMPROVED	WORSE	DEAD
20-30	2	0	2	3
30-40	3	1	6	3
40-50	3	1	8	1
50-60	1	0	2	3
60-70	1	0	2	0
	10 (23.8%)	2 (4.7%)	20 (47.6%)	10 (23.8%)

*Eight operative deaths and one recovery in which no spheres were inserted following entry of the cavity are excluded.

a wound which had opened as a result of tuberculous infection in the extrapleural space, one from a pulmonary hemorrhage after an operation for removal of the spheres, one from respiratory failure and progression of the disease, and one from a coronary occlusion.

Of the fifty-one cases, fourteen were considered to have definite bilateral cavitation while five others were suspected of having cavities on the opposite side. Of this group with bilateral cavities, only four had a bilateral procedure. One of these patients was considered improved (Case 4), one died, and the other two had persistent cavities under the spheres. Bilateral procedures were not done in the remainder, with the exception of one case, because of death or dyspnea. One patient diagnosed by laminography as having bilateral cavities had a unilateral procedure only (Case 1). The sputum converted and is reported to have remained negative since operation.

Eleven patients had had a previous thoracoplasty and one a bilateral thoracoplasty. Of these, seven had spheres inserted under the thoracoplasty. Two of these improved (Cases 1 and 2). Of the five who had spheres inserted on the opposite side, one improved (Case 10). Five of the twelve patients died and two became worse.

Of the six patients in whom we thought extrapleural pneumonolysis with plombage was indicated as the primary procedure, three improved, one had a tuberculous wound infection, and two died.

Bronchoscopy was done on six patients before operation and only one had findings suggestive of endobronchial tuberculosis. An attempt was made in our follow-up study, however, to do bronchoscopy on all patients who would consent. Of the eighteen patients on whom bronchoscopy was carried out, to date, twelve had ulcerative tuberculous tracheobronchial disease.

No effort has been made to draw conclusions from vital capacity studies. We have found the recorded figures to be inconsistent and frequently unreliable and have therefore excluded them from the study.

IMPROVED CASES

CASE 1.—J. B., a 49-year-old white man, had a bilateral three-rib thoracoplasty in 1942, followed by a revision on the right and removal of three more ribs in 1944. He was readmitted in July, 1945, with positive sputum. Planograms revealed bilateral cavitation and bilateral thoracoplasty. Extrapleural pneumonolysis with plombage was indicated. An anterior procedure was carried out on the right with insertion of fifteen spheres. Despite the fact that cavitation was present on the left, a bilateral procedure was not done since the sputum converted after the first operation. He has since been discharged from the sanatorium and is now working but has had no recent roentgenograms. The sputum is reported to be consistently negative on culture.

CASE 2.—C. L., a 45-year-old white man, had a two-stage, six-rib thoracoplasty in 1944. The cavity remained open. He was admitted to Duke Hospital in 1945 for revision. Classified as: right thoracoplasty with residual cavity, left F.A. A revision of the thoracoplasty was indicated. In July, 1945, anterior extrapleural pneumonolysis was performed under the thoracoplasty with insertion of sixteen spheres. Five months later, because of a persistent cavity, thirteen additional spheres were inserted through a posterior incision. Subsequent roentgenograms did not show a cavity and there was no progression of the disease. The pa-

tient had one positive sputum examination in March, 1947, but subsequent cultures were negative. The patient is now working as handy man in a Chicago hospital.

CASE 3.—W. H., a 42-year-old white man, was admitted to Duke Hospital in November, 1945, with cavitation on the right. Classified as: right F.A. with cavity, left M.A. A thoracoplasty was indicated. Through a posterior thoracoplasty incision the third and fourth ribs were removed, an apicolysis was performed, and an unknown number of spheres were inserted. The first two ribs were left intact in order to prevent deformity of the chest and to prevent pressure on the nerves and vessels by the spheres. A second stage was performed and the fifth, sixth, and seventh ribs were removed. The sputum was positive on discharge from the sanatorium in November, 1947, but subsequent specimens were negative on smear and culture. Clinically he is doing well. He refuses bronchoscopy.

CASE 4.—V. K., a 32-year-od white woman, was admitted to Duke Hospital in November, 1945, with roentgen evidence of bilateral cavitation. She had had one positive gastric washing in 1942, but had never had a positive sputum. Classified as: right F.A. with cavity, left M.A. with cavity. In this case we should have recommended thoracoplasty on the right with extrapleural pneumonolysis later on the left with paraffin or oil plombage. She had bilateral anterior procedures with the insertion of fifteen spheres on each side. Discharged from the sanatorium one year ago, she is now beginning to work. Bronchoscopy in February, 1948, revealed no endobronchial tuberculosis. Sputum has never been positive. Roentgenograms showed no progression.

CASE 5.—K. Y., a 61-year-old white woman, was admitted to Duke Hospital on Dec. 20, 1945, with cavitation in the right apex. Classified as: right F.A. with cavitation, left M.A. A thoracoplasty was indicated. An anterior procedure on the right was carried out with insertion of twenty-seven spheres. She improved clinically but is slightly dyspneic and continues to have a moderately productive cough. Single sputum specimens on two occasions were negative on culture.

CASE 6.—M. E., a 21-year-old white man, was admitted to Duke Hospital in February, 1946. Roentgenograms revealed cavitation in the right apex. Classified as: right M.A. with cavity, left M.A. A thoracoplasty was indicated. An anterior approach was done with insertion of forty-nine spheres. Subsequent roentgenograms showed no progression of the disease; there was no evident cavity. Sputum examinations have been consistently negative. The patient is now working and asymptomatic.

CASE 7.—C. P., a 21-year-old white woman, was admitted to Duke Hospital in February, 1946, for surgical collapse of a persistent cavity in the left apex. Classified as: right M.A., left F.A. with cavity. A thoracoplasty was indicated. An anterior procedure was done on the left with insertion of thirty-three spheres. She developed tuberculous meningitis eight months postoperatively, but gradually recovered with the aid of streptomycin. She is still on strict bed rest, but the sputum has been repeatedly negative on culture and symptomatically she is improved.

CASE 8.—E. M., a 42-year-old white woman, was admitted to Duke Hospital in February, 1946. Roentgenograms revealed a cavity with a fluid level in the right apex. Classified as: right F.A. with cavitation, left M.A. A thoracoplasty was indicated. An anterior procedure was performed, but the number of spheres inserted was not recorded. Sputum remained negative on smear and concentrate since discharge from the sanatorium one year ago and is now asymptomatic and doing regular housework.

CASE 9.—E. D., a 58-year-old white woman, was admitted to Duke Hospital in February, 1946, with cavitation on the right. Classified as: right F.A. with cavity; left clear. A thoracoplasty was indicated. An anterior procedure was performed with the insertion of forty-two spheres. Follow-up was done at the Florence-Darlington Sanatorium, Florence, S. C., and the patient is reported as "clinically well and sputum negative."

CASE 10.—P. M., a 31-year-old white man, had a two-stage thoracoplasty on the left in 1943, followed by an anterior stage in 1945. Roentgenograms on admission at Duke Hospital in May, 1946, revealed a cavity in the right apex. Classified as: right F.A. with cavity, left thoracoplasty. Planograms revealed no evident cavitation on the left. Extrapleural pneumonolysis with plombage was indicated. An anterior procedure was done on the right; seventy-two spheres were inserted. He had some dyspnea after operation. Planograms in December, 1947, revealed no evident cavitation. Sputum was negative on culture. The patient is now at home on limited exercise. He refuses bronchoscopy.

COMMENT

Extrapleural pneumonolysis with plombage is a desirable operation because it can be done in one stage, it is technically easy in the majority of cases, the patients tolerate it well, there is no deformity of the chest, the function of good lung tissue is maintained, and the hospital stay is short.

It is not surprising, therefore, that repeated attempts have been made to find the ideal material for insertion in the extrapleural space to maintain pulmonary collapse. It is not surprising also that these attempts have failed regardless of the type of material used, largely because of the susceptibility of the extrapleural space to infection; but of the many substances employed for this purpose, air,[3] oil, and paraffin[4] have been the most successful; their use, however, is limited to highly selected cases in which thoracoplasty is not indicated. Experimental studies at first seemed to indicate that Lucite spheres approached the ideal plombage substance because of their ease of introduction and their inertness in the body, but clinical trial has failed to substantiate the early promise of the operation.

Attention has been called in a previous report[1] to the dense hyaline tissue which forms around the spheres in the experimental animal. We found this to be true also in three clinical cases without preoperative evidence of infection in which we electively removed or attempted to remove the spheres from the extrapleural space. Since Lucite has been shown to be an inert material in the body the probable explanation for this phenomenon lies in the spherical shape of the plastic material which allows sufficient space between the spheres for serum to accumulate, clot, and organize. Once this dense hyaline mass has formed it is very difficult to remove the spheres it encases unless infection and liquefaction of the surrounding tissue occur. Removal of the spheres post mortem in one case required a mallet and chisel. It appears, therefore, that Lucite plombage in some cases must be a definitive procedure. If it is not successful in a given case, frequently no other procedure, such as thoracoplasty or resection, is feasible since the success of such an operation is dependent upon removal of the spheres.

Early results of the operation of Lucite plombage as performed at Duke Hospital[2] were encouraging but Wilson was careful to call attention to the fact that "too few cases had been completed and that too short a period of time had elapsed since operation to evaluate the results of this form of collapse therapy." The late results presented in our report emphasize the cogency of this warning when dealing with such a disease as tuberculosis.

Although our late results do not justify the continued use of Lucite spheres in the extrapleural space, it is possible that subsequent reports from other institutions may show that Lucite has, like paraffin and oil, a limited usefulness as a plombage material.

SUMMARY AND CONCLUSIONS

1. Fifty-one cases with operation a minimum of one year and six months to two years and ten months prior to this report have been presented in which extrapleural pneumonolysis with Lucite plombage was attempted or carried out. Ten patients (19.6 per cent) improved, three (5.8 per cent) did not improve, twenty (39.2 per cent) became worse, and eighteen (35.3 per cent) died.

2. Lucite plombage in some cases must be a definitive procedure, for once the spheres are inserted they are, in our experience, extremely difficult to remove unless the surrounding tissue is liquefied by infection.

3. The late results of the operation as performed at Duke Hospital do not justify continuation of the procedure.

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(See discussion on page 187.)

EXPERIMENTAL AND CLINICAL STUDY OF COLLAPSE THERAPY USING FIBERGLAS WOOL AND FABRIC

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COLLAPSE therapy is a well-established method of treatment in pulmonary tuberculosis. The simplest and most effective type of collapse therapy is artificial pneumothorax. Far from complete success is attained because of the high incidence of pleural adhesions. Even when they are completely severed by pneumonolysis, the lung may exhibit a tendency toward re-expansion. This is often initiated by the attendant pleural reaction which incites an obliterative process.

A selective type of collapse would seem to be the ideal, whereby only the diseased portion of the lung is collapsed. The undiseased portions are allowed to remain expanded in order most efficiently to carry on the functions of respiration.

True selective collapse is seen to occur in only a small percentage of cases and, at that, it is certainly not at the deliberate design of the pneumotherapist, but rather by virtue of the relative physiologic relationships existing between the several lobes. Selective collapse may be considered as occurring when the diseased lobe is in a state of peripheral atelectasis (associated with a relative bronchial hypotension), whereas, the remaining lung tissue exhibits a marked tendency to remain inflated.

When this selective condition does not prevail, collapse of the diseased areas also involves collapse of a part or all of the undiseased areas and thereby denies the patient a sizable portion of good respiratory tissue. Efforts to produce deliberate selective collapse have been made. Thoracoplasty has been used extensively with a considerable degree of safety, but this always results in a severe degree of physical deformity and the end results are not too gratifying.

Extrapleural pneumonolysis has been efficient in bringing about good anatomic selective collapse, but this procedure has been handicapped by a high incidence of space infection as well as some tendency toward re-expansion too early. Attempts to combat these complications have resulted in the introduction of a variety of space-filling agents in the belief that an obliterated space would be unfavorable for the development of infection.[1] Air, saline, oil, paraffin, body tissues, plain gauze, methyl methacrylate (Lucite), and others have been used.[2, 3, 4] Most of these agents are capable of maintaining the accomplished collapse, but each has demonstrated certain undesirable features which are invariably related to the physical, chemical, or physiologic properties of the agents used. In short, because of heaviness they produce pressure necrosis,

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soft agents tend to become liquefied, those that are unyielding result in penetration into lung parenchyma, irritating agents give rise to infection, and, as in the case of body tissues, these are too difficult to mobilize in sufficient enough quantities to be of value.

In the search for more suitable materials, two Fiberglas* products have been examined. One is a Fiberglas wool product grossly resembling raw cotton. The other is a woven fabric made from Fiberglas.

This is a material which is inorganic, nonallergenic, pliable, nonhygroscopic, noncombustible, and is light in weight, one cubic foot weighing 0.3 to 0.5 pound.†

In an attempt to evaluate the ability of animal tissues to tolerate these materials as foreign bodies over a period of time, two series of experiments were undertaken.

In one, Fiberglas wool was placed in the extrapleural space of each subject in a series of fifteen dogs. The dogs were subsequently sacrificed and specimens were taken from the implanted areas. The duration in time of these implantations ranged from one week to eight months. On gross examination, adhesions were usually noted occurring between the parietal and visceral pleura immediately adjacent to the implanted Fiberglas. Fifty per cent showed moderate to occasionally severe giant-cell foreign body reaction, and all showed either thickened pleura or fibrinous deposition, indicating considerable tissue reaction encountered in the use of raw Fiberglas wool.

The second group of experiments was carried out on fourteen dogs, in which Fiberglas fabric was applied directly to the intact pleural surfaces in the form of a bag to encompass a single lobe. First, the selected lobe was encouraged to become atelectatic, then, a sheet of Fiberglas fabric was fashioned about the lobe so as completely to contain it and prevent its re-expansion. Compression of the hilar structures was avoided. The dogs were sacrificed at intervals and specimens were obtained at ½, 1, 4, 5, and 6 months. Upon gross examination, the Fiberglas covering was found in place, undisturbed, showing no adherence to the parietal pleura. The cloth was held in close apposition to the visceral pleura by an adherence which was very fine and permitted extreme ease in removal by peeling, leaving a clean, shiny pleural surface, even at 6 months. In five unselected cases, efforts at re-expansion of these lobes were immediately successful by applying positive pressure through the dog's endotracheal tube. On microscopic sectioning of the remaining nine of these lobes, there was seen in all cases an excellent atelectasis with patent bronchi. It was usual to see a minimal mononuclear infiltration and a very mild pleural reaction. The absence of fibrosis in even the oldest specimens is indicative of a reversible condition.

*Fiberglas; T. M. Reg. U. S. Pat. Off. OCF Corp.

†The Fiberglas superfine wool is composed of glass fibers random-oriented in a batt. Fiber diameter averages .00006 inch. The fabric, known as "116 cloth," is .004 inch thick and is of balanced Fiberglas 450-½ yarns, 60 ends per inch in warp and 58 per inch in the fill. Each strand is formed of approximately 200 individual filaments. The average filament diameter is approximately .00022 inch.

Late postoperative x-ray studies were made on five dogs and showed good collapse of the treated lobes and normal x-ray appearance of the untreated lobes.

Comparable procedures were carried out on a series of patients in order to achieve selective collapse.

CASE REPORTS

CASE 1.—N. W., a 26-year-old white woman, was admitted May 13, 1948, with a history of known pulmonary tuberculosis since June, 1943. Pneumothorax was instituted on the left at that time and the patient became symptom-free until September, 1947, at which time x-ray showed development of tuberculous involvement in the right upper lobe. Bed rest was instituted and the lesion became more extensive; a cough developed during the past six weeks. Physical examination revealed a thin, 85 pound, white woman in no distress and presenting findings of pneumothorax on the left. Chest roentgenogram demonstrated the left partial pneumothorax and also tuberculous infiltration of the right upper lobe with the presence of a central area of cavitation. On May 14, 1948, through the fifth rib bed an open pneumonolysis with Fiberglas covering was done. The patient made a rapid postoperative recovery, and smears on three occasions for acid-fast bacilli were negative.

CASE 2.—J. D., a 28-year-old white man, was admitted Oct. 13, 1947, a diagnosis of pulmonary tuberculosis having been made in 1943. The patient received right pneumothorax in 1946 and this was shortly discontinued as ineffectual. Examination revealed a fairly well-developed and well-nourished young man in no distress. There were absence of breath sounds below the fourth rib on the right, and crepitant râles were heard in the area of the scapula. X-ray showed infiltration with cavitation in right upper lobe, and free fluid in the right base. On Oct. 22, 1947, an open pneumonolysis was done through the right fifth rib bed and the upper lobe was encompassed in Fiberglas fabric. The patient made a rapid postoperative recovery and the sputum was converted to negative.

CASE 3.—H. H., a 34-year-old white man, was admitted Feb. 26, 1948, having been ill since 1943; a diagnosis of pulmonary tuberculosis had been established in 1947. Pneumoperitoneum was instituted in February, 1947, because attempts at pneumothorax were unsuccessful. The patient had 33 hemoptyses in early 1948 and was admitted in that state. Examination revealed a thin, pale, young, white man in no great distress. There were râles over both anterior apices and left posterior upper lung fields and a friction rub was heard over the latter. X-ray showed pneumoperitoneum and dense infiltration in the left upper lobe with several cavities in evidence. On Feb. 27, 1948, an open pneumonolysis was done. The upper lobe resisted satisfactory collapse because of the caseous pneumonic infiltrative process resident in the parenchyma. Collapse of the lobe was about 60 per cent, nevertheless the lobe was wrapped with Fibergas fabric and the remaining two lobes were allowed to re-expand to fill the residual space. The patient made a rapid immediate postoperative recovery, had no recurrence of hemoptyses, and gained ten pounds in weight in the following three months. The sputum, however, continued positive. This type of patient represents a poor choice for any type of collapse therapy, and should rather have been allowed resection of the lobe.

CASE 4.—W. B., a 36-year-old white man, was admitted Jan. 15, 1947, having had a diagnosis of pulmonary tuberculosis four years prior to admission. Pneumothorax had been unsuccessfully attempted on the right. Physical examination revealed a fairly well-developed and nourished white man in no distress. X-ray revealed infiltration in the right upper lobe with cavitation, and also a dense infiltration in the left hilar region. On Jan. 17, 1947, a right extrapleural plombage was done by first lining the extrapleural space with Fiberglas fabric and then filling the resultant fabric sac with Fiberglas wool. This modification over the animal experimentation was done to protect the surrounding tissues from the irritation found to be caused by the raw or uncovered Fiberglas wool.

The patient made a rapid postoperative recovery and, although the sputum is still positive, the Gaffky count is lower and there is seen on x-ray considerable clearing of the contralateral disease. The patient is asymptomatic.

CASE 5.—J. P., a 31-year-old white man, was admitted on Oct. 24, 1947, for check-up relating to a marked increase in dyspnea and the increasing tendency to turn a blue color with exertion. Past history revealed that he had been a "sick baby" at birth, and that he had always been troubled with shortness of breath since he could remember. In recent months he had noted swelling of the ankles when doing limited light work. Physical examination showed a well-nourished and developed young man in no distress when resting. There was a marked blue cyanosis of the lips, tongue, and nail beds. There were no râles heard in the chest. The heart was enlarged and there was a systolic thrill over the entire precordium. There was a loud, grade 4, systolic murmur present. Respiratory function tests, angiocardiography, and routine x-rays strongly intimated the diagnosis of (1) pulmonary tuberculosis, bilateral upper lobe, and (2) aplasia of the right ventricle, associated with hypoplasia of the pulmonary artery, and double superior vena cava. Therefore, reasoning that a single stage operation should be sufficient to afford major relief for both the pulmonary disease and also the cardiovascular anomaly, on Dec. 19, 1947, an open pneumonolysis was done through the fifth rib on the right and a Blalock operation was done, anastomosing the right subclavian artery to the hypoplastic right pulmonary artery. The patient's immediate postoperative course was excellent. Coughing, straining, or walking no longer produced the extreme blue cyanosis—it was no longer present. However, due to bronchial hypertension, associated now with pulmonary arterial hypertension, it very soon became impossible to keep the upper lobe collapsed as was intended at the time of operation in order to control the pulmonary disease on the worse side. Therefore, on Jan. 16, 1948, an extrapleural pneumonolysis was accomplished, the space lined with Fiberglas fabric to protect the surrounding tissues, the sac filled with Fiberglas wool, and the wound closed in the usual manner with good healing. The sputum remained positive, not surprising in view of the active disease in the contralateral side. The patient was referred to a local sanatorium for a regimen of medical therapy.

CASE 6.—M. W., a 45-year-old white woman, was admitted on March 13, 1948, known to have had pulmonary tuberculosis since 1945. The patient had been on routine sanatorium care and because of the extent and nature of the lesions, intrapleural pneumothorax was judged as impossible, and she was admitted for surgical treatment. On examination there was seen a well-developed, obese (188 pounds), middle-aged woman, looking much older than her stated years, but in no distress. X-ray showed bilateral upper lobe chronic fibroid tuberculosis with multiple cavitation bilaterally, more extensive on the right. There were persistent râles heard over the left upper lung fields. On March 19, 1948, an extrapleural pneumonolysis was done through the right fourth rib and the space lined with Fiberglas fabric and then filled with Fiberglas wool, as was done in the previous cases. Table I shows the pulmonary function studies before and after this operation. The patient made an uneventful recovery, except that there was noted a too great collapse of the lung due to overfilling of the space. Thus, on April 9, 1948, the old scar was incised down to the neck of the Fiberglas

TABLE I. M. W., 45 WF

	ADMISSION	APRIL 8, 1948	MAY 8, 1948
Actual vital capacity	2,550.0 c.c.		1,250.0 c.c.
Respiratory rate (per min.)	11.0	13.0	12.0
Tidal air (liters)	0.609	0.490	0.417
Resting minute ventilation	6.7	6.3	5.01
Maximal breathing capacity	43.2	45.9	29.54
Ornsteen factor	6.4	7.2	5.8
Oxygen rebreathing bag	10.5		10.1
Carbon dioxide rebreathing bag	5.0		7.62

sac, the sac was opened, and about one-half of the previously inserted glass wool was removed. At the same time, a first stage thoracoplasty with removal of the first and second ribs was carried out on the left (contralateral) side. The patient made an uneventful recovery, so, on April 28, 1948, the thoracoplasty was completed with the removal of the third, fourth, and fifth ribs. Again, recovery was uncomplicated and Table I gives the pulmonary function figures. The patient showed no more dyspnea at discharge than she did on admission. The sputum was converted to negative.

CASE 7.—J. G., a 38-year-old man, was admitted April 9, 1948, dating his illness from 1937. A diagnosis of pulmonary tuberculosis had been established in 1938. Pneumothorax was instituted on the left. In 1941 a six-rib thoracoplasty had been done. He was admitted for surgical treatment of a reactivated lesion in the left upper lobe. Examination revealed a well-developed and nourished young man in no distress. There was evidence of a right thoracoplasty with a well-controlled underlying lung lesion. There were persistent râles heard through the left upper lung fields. X-ray showed a right thoracoplasty with no evidence of tuberculous activity. The left lung field showed evidence of a thickened pleura with tuberculous infiltration in the upper lobe. On April 17, 1948, an extrapleural pneumonolysis was done on the left. The space was lined with Fiberglas fabric and the sac filled with Fiberglas wool, in the same manner as the preceding cases. The patient made an uncomplicated and rapid recovery, and it is too early to evaluate the sputum conversion.

CASE 8.—T. C., a 59-year-old white man, was admitted Sept. 22, 1947, his third admission. A diagnosis of pulmonary tuberculosis had been made in 1938. In November, 1946, a thoracoplasty had been done on the right side. This was unsatisfactory so in February, 1947, a revision was done with the introduction of raw Fiberglas wool into the extrapleural space in the hope of gaining a better collapse. It is noteworthy that this is the only case in which the Fiberglas wool was not covered with Fiberglas fabric to protect the tissues. Soon there developed a draining and infected sinus in the revision scar. In the meantime, several attempts at pneumothorax on the left were made without success. Physical examination revealed an emaciated white man in no acute distress. There was bronchial breathing in the left middle lung fields. The right posterior thorax showed a well-healed thoracoplasty revision scar with a small draining sinus. X-ray revealed a right thoracoplasty with no apparent active disease in the right lower lobe. There were mottled densities in the left upper lobe suggestive of cavitation. On Sept. 24, 1947, the sinus tract was excised along with the previously deposited raw Fiberglas wool, which was now in the form of a fibrofatty tissue mass measuring 20 by 10 by 2.5 cm. On microscopic section there was seen extreme fibrosis with many foreign body giant cells, which contained colorless fibrils. The wound healed per primam and the patient was discharged to sanatorium care.

CASE 9.—H. I., a 34-year-old white man, was admitted April 1, 1947, pulmonary tuberculosis having been diagnosed in 1945. Pneumothorax was attempted without success on the right. Examination revealed a well-developed and nourished man in no distress. There were râles in the right upper lung fields. On April 2, 1947, an extrapleural pneumonolysis was done on the right and the space was lined with Fiberglas fabric; this sac was filled with Fiberglas wool. The patient made a rapid recovery and the sputum was converted to negative and has so remained to the present.

CASE 10.—R. S., a forty-three-year-old white woman, was admitted May 12, 1948, for surgical treatment of bilateral pulmonary tuberculosis, known as present since 1928. The patient had been treated in a number of institutions, experiencing many remissions and exacerbations. Bilateral pneumothorax, right phrenemphraxis, and pneumoperitoneum were all abandoned as unsuccessful. On physical examination, the patient was well developed and nourished and presented râles over both upper lung fields with percussion dullness in the extreme apices. Chest roentgenograms showed upper lobe involvement with suggestion of bilateral cavitation. On May 19, 1948, extrapleural pneumonolysis was done on the left, with instillation of Fiberglas fabric lining packed with Fiberglas wool in the usual manner. The

postoperative course was uneventful. It was contemplated to do the opposite side within a two-week period.

TECHNIQUE

For both types of operations the patients are given a general anesthetic using the endotracheal tube and placed in the lateral horizontal position. A curved periscapular incision is made and carried through the superficial back muscles to expose the costal cage. With the scapula translated superoanteriorly, a section of the fourth or fifth rib is removed. When the use of Fiberglas cloth wrapping is intended, the pleura is opened and an intrapleural open pneumonolysis is done. The lobe is rendered atelectatic and a sufficient amount of cloth is cut to allow complete investment of the collapsed lobe. The free edge or periphery is infolded and sutured around the circumference of the lobar hilum with interrupted No. 40 cotton. This is accomplished in a gathering manner so as to provide a snug but not tightly constricting covering.

When the use of plombage is desired, an extrapleural stripping is carried out in the standard manner. Then, the space so created is completely lined with one layer of Fiberglas fabric with the neck of the cloth sac presenting at the wound. This sac is then filled without resorting to tight packing and the neck of the sac is sutured shut. In both operations a drainage tube is left in place with suction for twenty-four to forty-eight hours to prevent accumulation of fluid incident to the operative trauma. The patients have all been ambulatory within twelve to twenty-four hours, restricted only by the limits of the tubing.

DISCUSSION

The indications for these procedures would seem to be the same as for any type of lesion in which one would expect favorable response to permanent collapse therapy of a selective nature. The problem of overdistention of the uninvolved lung tissue might give concern in the use of the Fiberglas wrapping. This concern, however, should not be any greater than that appreciated in the normally occurring sequence of pathologic changes seen when lesions of any type heal. This healing is attended by fibrosis and sometimes extensive contraction which always lays open the contiguous normal lung tissue to compensatory emphysematous changes.

It is too early to evaluate the proper placement of these procedures in the treatment of tuberculosis. Other techniques have made their entry showing good promise, only to fail due to certain inherent faults, which in the course of time precipitated late complications. Caution is therefore advocated in the use of these procedures until more evidence is at hand concerning the limits of usefulness and the development of possible complications. Encouragement is gained from the early results and it is hoped that a continuing favorable follow-up will justify extension of this project.

SUMMARY

Experimental studies were undertaken using a combined series of twenty-nine dogs in which two procedures were done. In one, extrapleural Fiberglas raw wool packing was affected. This resulted in tissue reaction which was

regarded as unsatisfactory. In the second series, Fiberglas fabric was used intrapleurally to invest a selected lobe. This was found satisfactory in so far as excellent selective atelectasis was accomplished, and also that the implantation of this material occasioned only a minimal tissue reaction. The case histories of ten patients were reviewed. In three, the upper lobes were wrapped in Fiberglas fabric to accomplish selective collapse. Seven had extrapleural pneumonolysis and filling of the created space with Fiberglas wool. One of these had the raw wool alone placed in the space and this resulted in the same kind of tissue reaction as seen in the dogs in which there was considerable foreign body response, and this became infected and had to be removed. The remaining six had the spaces first lined with Fiberglas fabric which was shown in the dogs to be practically nonreactive, and then the lined space was filled with the raw Fiberglas wool. No infections resulted in these and the collapse was regarded as satisfactory.

We wish to express our thanks to the Owens Corning Fiberglas Corporation, for their assistance, and to Dr. G. F. Froio for his work on the pathologic studies. We are also indebted to Dr. Martin Entine who first suggested the use of Fiberglas as a prosthetic agent.

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DISCUSSION ON "THE CLINICAL USE OF A PROSTHESIS TO PREVENT OVERDISTENTION OF THE REMAINING LUNG FOLLOWING PNEUMONECTOMY" BY DR. JULIAN JOHNSON, DR. CHARLES K. KIRBY, DR. C. S. LAZATIN, AND DR. J. A. COCKE*; "AN EVALUATION OF EXTRAPLEURAL PNEUMONOLYSIS WITH LUCITE PLOMBAGE," BY DR. JOSIAH C. TRENT, DR. JAMES D. MOODY, AND DR. JOSEPH S. HIATT, JR.†; AND "EXPERIMENTAL AND CLINICAL STUDY OF COLLAPSE THERAPY USING FIBERGLAS WOOL AND FABRIC" BY DR. THOMAS J. E. O'NEILL, DR. HECTOR P. REDONDO RAMIREZ, AND DR. ROBERT G. TROUT

DR. WILLIAM R. RIENHOFF, JR., Baltimore.—I have enjoyed the papers very much indeed, but there are two points I would like to comment on. In the first place, I think the term "compensatory emphysema" is an inappropriate one and used rather loosely. Emphysema is a pathologic condition which denotes fragility of the elastic tissue in the alveolar septa, together with rupture of alveolar walls and confluence of alveoli. This is not a compensatory mechanism but a pathologic change. What is meant, and the correct term seems to me to be used, is "compensatory dilatation" of the remaining lung following total pneumonectomy. In 1935,‡ I believe, in conjunction with Heuer and Reichert, a series of experiments were performed to prove what happened to the remaining lung following total pneumonectomy in the dog. This experimental work showed conclusively that the remaining lung undergoes a compensatory dilatation following removal of one lung which dilatation is confined to the definitive respiratory unit, composed of the ductus alveolaris, atria, and alveoli, there being no changes in the main bronchi and not the slightest evidence of the pathologic condition emphysema, as just defined.

*See page 164 for article by Johnson, Kirby, Lazatin, and Cocke.
†See page 173 for article by Trent, Moody, and Hiatt.
‡Rienhoff, William F., Jr., Reichert, Fred L., and Heuer, George J.: Bull. Johns Hopkins Hosp. **57**: 373-383, 1935.

In various autopsies, including the first case done at the Johns Hopkins Hospital by me in 1933 for fibrosarcoma of the lung (the patient drowned five years following operation), and in another instance, a woman of 70, who died six months after operation due to cerebral metastases, section of the lung at autopsy showed no evidence whatsoever of emphysema, but established unquestionably the compensatory dilatation that was found to exist experimentally. Also, there has never been any evidence of right ventricular hypertrophy following total pneumonectomy. Therefore, I think the term "compensatory emphysema" should not be used, but instead, "compensatory dilatation."

The second point that interested me particularly in Dr. Johnson's remarks was the fact that Lucite balls were used to obliterate the thoracic cavity following total pneumonectomy for malignant tumors. It is my opinion that compensatory dilatation of the remaining lung following removal of one lung for malignant tumor should not be prevented, therefore, the use of plombage is contraindicated. This may not, and probably does not, apply to total pneumonectomy for tuberculosis, because dilatation of the remaining lung might lead to a tendency to spread of the disease in that remaining lung. However, that is not true of compensatory dilatation in the remaining lung following total pneumonectomy for nonspecific infections such as diffuse bronchiectasis or infected multilocular cysts. In a series of more than 200 cases of total pneumonectomy, mainly for malignant tumor, but also for inflammatory conditions, we have not found it necessary to use any form of intrathoracic plombage and have felt that the reason our patients have had a minimal amount of impaired cardiorespiratory function is that there was a maximum opportunity for the remaining lung to undergo compensatory dilatation. The follow-up study at the present time is being continued and will be reported at a later date.

The very fact that after operation patients having a total pneumonectomy for one reason or other are somewhat short of breath even though in an oxygen tent, but later are able to breathe perfectly well and not only perform their vocations but as well their avocations, would seem to indicate that compensatory dilatation is physiologically very helpful rather than harmful. Therefore, any method employed to discourage or impair this compensatory dilatation, it seems to me, would have a deleterious effect upon the patient.

DR. H. R. DECKER, Pittsburgh.—I enjoyed these papers very much. I have had no experience with the new products used as packs after extrapleural pneumonolysis but I have had some experience with paraffin pack, a rather small series to be sure, but interesting to me because I was able to trace the cases through a period of from eight to fifteen years. In a group of 16 cases in which I used paraffin pack, for apical cavitation and bilateral tuberculosis, there are 3 patients surviving now for periods of eight, thirteen, and fifteen years, and apparently perfectly well. I will show later the slides of these cases and discuss them a little more fully. I have a feeling from hearing the paper by Dr. Trent and his co-workers that they have discarded Lucite as the medium for holding the extrapleural pneumonolysis down. I am sure the figures published through the literature with reference to paraffin pack are better. The difficulty, however, is this: Almost all figures that one reads in the literature cover a follow-up period of not more than five years. Most of them are just for immediate postoperative periods or less than three years. I am sure, therefore, that the figures as published will deteriorate as time goes on. It is so in my own small series. For instance, the series of 16 patients, in which the pack was used for tuberculosis, shows only 3 known to be living and well. Eleven are known to be dead over a period of from one to four years following the use of the pack. The cause of death mainly is progressive tuberculosis.

I would like to show two or three slides of these patients who are surviving today after the use of the pack.

(Slide 1.) The patient was not in very good physical condition and we suggested this procedure. This is the picture taken immediately after the operation.*

(Slide 2.) The man was admitted to my care following persistent hemorrhage in 1933. It seemed that the paraffin packing might be sufficient to stop the hemorrhage, and it did.

*The second slide shows the pack after eight years, the apical cavity closed. The patient is clinically well of tuberculosis.

This patient was discharged in 1937 as being well. This picture was taken two months ago. He worked four years in Czechoslovakia on his farm and then as a prisoner in a German agricultural camp. He is now on his way to a farm in Oregon, apparently entirely recovered and having no symptoms from the pack still in place.

DR. J. B. GROW, Denver.—I was very impressed with these papers, although when we consider the operation of extrapleural pneumonolysis we must consider an operation which has not found a great place in treatment of tuberculosis. At the same time there are many instances in which there is no other procedure to be offered the patient. These were well outlined by Dr. Trent. There is no real competition between thoracoplasty and plombage in the patient whose condition is suitable for thoracoplsay. However, for the patient not suitable for thoracoplasty, plombage offers the only procedure which will satisfactorily control the disease. As Dr. Trent said, the advantages of plombage are: (1) Any portion of the complete lung may be collapsed. (2) Pulmonary function is preserved. (3) A one-stage procedure is done instead of multiple procedures. (4) The economic factors, hospitalization and expense, are reduced. (5) Following plombage there is no paradoxical motion and a decreased incidence of spread. (6) Last, and least important, plombage is not deforming.

Our experience with plombage is based on 53 cases at the National Jewish Hospital in Denver, and 26 private cases. The cases at the National Jewish Hospital are better followed, and those are the ones I will present. Of the 53 cases, 78 per cent of those over six months following operation have a negative sputum. There was one death, which occurred in a patient who had a congenital heart ailment, a high blood count, and resulted from cerebral thrombosis.

I think it is important in considering the subject, to consider the technical features, because apparently the results are largely dependent on two factors. First, the selection of cases, and second, the technique of operation.

(Slides.) The anterior approach as described by Graham and Joannides is used. An incision is made along the anterior axillary line; the second rib is resected and the cleavage plane established between endothoracic fascia and chest wall. From an anterior approach, since most tuberculous cavities are posterior, one can dissect the lung from the chest wall under direct vision, in the adherent area. The importance of thorough dissection of the mediastinum should be emphasized. As Dr. Trent pointed out, extrapleural plombage may have to be a definitive procedure unless the lung can be dissected away from the mediastinum. When the pneumonolysis includes stripping of the upper lobe from the mediastinum, it is possible to do a lobectomy or some other type of resection under the Lucite plombage, leaving the Lucite in place.

The next patient shown had a rather thorough pulmonary function study. In spite of advanced disease he had a 124 liter per minute maximum breathing capacity, which is considered quite good. Following bilateral plombage, his maximum breathing capacity was 114 liters per minute, a very slight reduction. He still has a positive sputum and is being considered for resection of the right upper lobe under the Lucite.

The next patient was a social worker for the hospital. Following a first-stage thoracoplasty there was suppurative bronchitis and spread of the disease to the lower lobe. The entire lung became atelectatic and, in spite of repeated attempts at bronchoscopic aspiration and re-aeration of the lung, the lung remained collapsed. Normal weight was 132 pounds; she was down to 82 pounds and slipping very fast in spite of streptomycin. With the experience of these previous cases it was thought possible to carry out a rather extensive procedure on a desperate risk patient. An extrapleural pneumonolysis of the entire lung was done in one stage.

To the present time, four lobectomies and one pneumonectomy have been performd subsequent to extrapleural pneumonolysis and Lucite plombage. Pulmonary resection leaving the plombage material in place is a technically satisfactory procedure. If extrapleural pneumonolysis is done, it is important to strip the lung from the mediastinum in order to make subsequent resection possible in those patients in whom the disease is not controlled by the collapse therapy.

DR. O. C. BRANTIGAN, Baltimore.—Following the work of Dr. D. Wilson and Dr. J. Johnson I have been very much interested in Lucite ball plombage in the treatment of pulmonary tuberculosis and Lucite ball prosthesis after pneumonectomy.

The report on Lucite ball plombage by Dr. Trent is extremely discouraging. It is difficult to justify his early morbidity and mortality but the late results are difficult to challenge with our present state of knowledge. I now have completed plombage on 29 patients suffering from pulmonary tuberculosis. There have been no complications nor untoward effects. The first operation was done about one year ago. I do not have statistics on end results since it is too early to consider seriously the end results.

I vary with Dr. John Grow only on technical points. The posterior approach is preferred rather than the anterior or lateral. The apex of the lung should not be freed from the mediastinum. If left adherent to the mediastinum there will be better drainage of the bronchi and the disease thus cannot be displaced into good lung tissue and remain uncollapsed.

In considering Lucite ball prosthesis after pneumonectomy, or thoracoplasty after pneumonectomy, there are two distinct problems—one is the cardiorespiratory function and the other is the flare-up of tuberculosis after pulmonary resection for tuberculosis. For patients who have had pneumonectomy for nontuberculous disease, as advocated by Dr. Rienhoff, I have done neither thoracoplasty nor prosthesis and the results have been entirely satisfactory. If cardiorespiratory difficulty arises one can do a thoracoplasty or prosthesis. In patients who have had pneumonectomy for tuberculosis the problem is different. If one waits for a flare-up of the disease, it is usually too late for treatment. An adequate thoracoplasty after pneumonectomy is a formidable procedure. In the last ten patients, through a small incision, under local anesthesia, Lucite ball prosthesis has been done four to ten weeks after pneumonectomy. The fifth such patient developed a *Staphylococcus aureus* empyema and subsequently died. The remaining nine patients have had no difficulty, but the follow-up is short since the first was done in October, 1947.

DR. STUART W. HARRINGTON, Rochester, Minn.—I am very reluctant, and am certain most of us are, to put a foreign body into the chest cavity or any part of the body, as a matter of fact, where it is to be permanent. However, there are cases in which the remaining chest cavity will require thoracoplasty, and I think it advisable that we explore these means by which we may avoid thoracoplasty to avoid overdistention or mediastinal shift which may be troublesome to the patient.

My associate, Dr. Clagett, has been particularly interested in developing a prostheses for the chest cavity following pneumonectomy. He has given me a résumé of the work he has done in this field to the present time, which, with your permission, I will read:

"Recently, Dr. Small, Dr. Grindlay, and I reported on the use of gelatin foam sponges to fill the pleural cavity after pneumonectomy. We have carried out extensive studies on animals and have used the material clinically in 20 pneumonectomies. We believe this material has considerable merit, but it is gradually absorbed and since it causes no tissue reaction or fixation, does not accomplish any permanent benefit. Experience with plastic polythene in various forms such as tubes for replacement of defects after tracheal resection, tubes for repair of stricture of the common duct, and sheets for replacement of dural defects, has demonstrated that this material causes little tissue reaction, is elastic and flexible, and can be molded or worked into various forms and shapes. We were encouraged to try the use of this material for prostheses that could be used to occupy the pleural space left after pulmonary resection. Bags, roughly lung shaped, were made of this material and were loosely filled with Pyrex glass fiber. This resulted in a soft, flexible, and very lightweight prosthesis, which has some elasticity but which will retain its shape. Such bags were placed in the empty pleural cavity after pneumonectomy in a series of dogs. Neither mediastinal shift nor pleural effusion has occurred in these animals. Pleural reaction consists of only a slight degree of thickening. Roentgenographically these bags are almost invisible.

There have been no indications of any physiologic disturbance. The postoperative course and condition were much better than in the control dogs which underwent pneumonectomy without insertion of the prosthesis. Exploratory thoracotomy two and three months after pneumonectomy has shown only slight thickening of the pleura and at most, only a few centimeters of clear sterile fluid.

"This study on dogs has been in progress only a few months, and while it has been going on, another series of experiments has been carried out in rats. The rats have had one lung removed and the empty space filled with a prostheses. These rats have been killed at various times postoperatively, and the pleural reactions studied. Briefly, much less fluid and exudate than usually follows pneumonectomy was found, and this fluid contains fewer cells; the pleural cavity surface and the bag are early surrounded by a soft membrane of fiber. At first the membrane is relatively thick, but within two months it is only a thin layer of fiber. The tissue is not adherent to the bag; the bag can be removed without difficulty.

"There are some technical difficulties in the manufacture of these bags in the desired shapes and sizes. We hope to be able to develop sizes and shapes to correspond to the various segments of lung as well as the entire lung, in the near future. If these prostheses work out in long-term studies on animals as well as they have thus far, we believe such prostheses may have a useful place in thoracic surgery."

As I have said, we all hesitate to use prostheses but there are occasions in which I think they may have to be used, in order to avoid thoracoplasty.

DR. A. M. VINEBERG, Montreal.—I should like to draw your attention to a procedure which we are now using at the Grace Dart Hospital, Montreal, which we think is worth while because we use a living fascial suture, or shall we say a living fascial sheet. We take a fascial sheet from the leg and sew it across the chest after depressing the apex of the lung as is done in extrapleural pneumothorax. We suture it to the fifth interspace and on the mediastinal side we suture the fascia to the lung on its mediastinal side. In this way we hope to keep the apex of the lung down, and in the few cases in which we have used this treatment, it has been successful. I should like to show you one example of this procedure.

In subsequent cases we have placed the fascial sheet directly on the apex, and in this way have produced what nature often produces, a thickened part over the lung which we wish to collapse.

DR. LYMAN BREWER, Los Angeles.—I should like to report briefly on a small series of cases in which Dr. Dolley and I have used the Lucite ball plombage with a very limited indication. We have not employed Lucite balls as a primary plombage in place of extrapleural pneumothorax, but have reserved this technique for bad risk cases where thoracoplasty had failed. In these patients although pulmonary resection might have converted the sputum, such an operation was not possible due to contralateral disease or poor general condition. This includes a group of 31 patients, over 35 years of age, who had vital capacities around 1,000 to 1,500; five had severe diabetes. No attempt was made to pick ideal cases as we were interested to see what this procedure would do. These patients have been treated in the period from the past three to fifteen months.

The technique of operation was simple. The operative thoracoplasty wound was opened, a small extrapleural space was developed, and the Lucite balls were placed in this space. We did not free the mediastinum as has been recommended earlier this morning. It is really too early to consider the results in these cases, but we have had 15 negative cultures. In analyzing our failures, we find that bronchial stenosis, tuberculous tracheobronchial disease, and cavity elsewhere in the lung have been mainly responsible for failure to convert the sputum. In addition we have gained the preliminary impression that certain giant tension cavities, and lobes almost totally destroyed by tuberculous disease, are not suitable for extrapleural Lucite pack following unsuccessful thoracoplasty. In some cases, too, it was impossible to get adequate separation of the lung. (Slides.)

The majority of the cases reported this morning, I believe, have been mainly ones in which the Lucite ball plombage has been used primarily in place of extrapleural pneumothorax. This represents a group of patients in whom the pulmonary disease was not as well stabilized as those in our group in which considerable fibrosis of the lung was present after thoracoplastic collapse. It seems to be a reasonable preliminary conclusion that the lung in which exudative disease is present with little pulmonary fibrosis does not tolerate well an extrapleural Lucite ball plombage, while the more stable fibrotic lung following thoracoplasty will tolerate such a plombage.

We believe, therefore, in cases where pulmonary resection is not feasible following unsuccessful thoracoplasty, further trial of extrapleural Lucite plombage is definitely indicated.

DR. C. W. LESTER, New York.—It has been my privilege to have been associated with Dr. André Cournand and his staff in the study of pulmonary function after pneumonectomy, particularly in children. It is our feeling that overdistention is a condition to be reckoned with and should be avoided. Our first studies one or two years after operation made us think that perhaps overdistention was not a factor where children were concerned. Several years later we restudied the same patients after they had passed the age of puberty and had become adults, and we found overdistention was something to consider and that pulmonary function was definitely less good in those who had distention than in those who did not. We therefore thought that some method should be attempted to prevent distention of the lung while these children were growing. Thoracoplasty, in a child, of course, is a very deforming operation. We had not thought of Lucite, but of course most of the operations that I have done were in infected cases. Here suppurative lungs are being dealt with, and infection in the presence of a foreign body is something to be avoided. We therefore have tried a modified form of thoracoplasty which really is not a thoracoplasty at all. In opening the chest the fifth rib is removed completely, and then the fourth and sixth are removed in their posterior halves. What we hope to obtain by this is that, as the child grows, the upper part of the chest will contract, and the rising diaphragm will prevent overdistention at least until the child reaches adult life when a thoracoplasty can be accomplished without so much deformity. We are, of course, studying these children as we go along.

(Slides.) These two cases are very recent. We cannot draw conclusions from them as yet. One was an 8-year-old who had extensive bronchiectasis of the left lung with contracture of the lung and shift of the right chest into the left side. After operation in which the fifth rib can be seen resected completely and the fourth and fifth partly resected, the heart has moved back into the right side of the chest and there is much less distention. This child was too young to attempt functional study; oxygen saturation was normal in the blood.

The other child had essentially the same condition. After operation some shift can be seen, not very much. We found on functional study the residual air was 25 per cent, which is essentially normal, and the results have been good so far.

DR. J. D. MURPHY, Oteen.—When a stone is dropped into the placid surface of a millstream, there is set in motion a series of centrifugal waves which ultimately cover the whole pond. So, when the first Lucite ball was dropped into the extrapleural space at Duke University, Oteen being geographically close to Duke was early in contact with this wave of patient enthusiasm and we succumbed. Having had some unfavorable experiences during the extrapleural pneumothorax era of 1938 to 1939, we determined to screen our cases carefully and attempted to select, therefore, only those cases we decided were good thoracoplasty risks.

(Slides.) I will give you briefly our experience. There were 22 cases in all; 14 primary operations and 8 operations following thoracoplasty in which the cavity had not been closed. There was sputum conversion in 27.7 per cent, which does not approach that of thoracoplasty. There were 3 empyemas and 3 fistulas in the primary group; 1 empyema in the secondary cases and in 2 patients migration of the balls. In 1 patient 5 balls migrated into the mediastinum. They are still there and we are unable to find them. In one patient a ball migrated up to the neck and produced pressure on the brachial plexus and subclavian arteries. That

was removed. We have removed a total of 9 packs. In cases in which empyema follows and the material is liquefied, it is not difficult. In cases in which there has not been empyema each ball is enclosed in a fibrous capsule, presenting a honeycomb-like structure. These are more difficult to work with, but can be removed without too much trouble. A uterine tenaculum forceps has been used to remove several deeply embedded balls. We have had no mortality. We started these operations in September, 1946, and became so discouraged by 1947 that in June we discontinued them. We will wait awhile before other plombage materials are tried.

DR. FRANK S. DOLLEY, Los Angeles.—It has become evident in listening to the several uncorrelated papers that have just been delivered on the subject of the introduction of Lucite balls between the lung and the chest wall for the local collapse of lung tissue affected by tuberculosis, that some attempt should be made to evaluate the things that have been said of this particular form of treatment.

My associate, Dr. Brewer, has just reviewed our results in the use of the Lucite pack. From these results we are more than ever encouraged in the use of Lucite balls. In other papers the operation has been uniformly condemned. The reason for this divergence in opinion is due, I am certain, to the selection of cases.

In all our cases except one, we have established a Lucite pack following thoracoplasty. We resect a small portion of the rib immediately beneath the thoracoplastic area, and find the line of cleavage at that point. After a small pocket is obtained by digital dissection, the space is enlarged, under direct vision by use of a Cameron light. By sharp and blunt dissection the lung and parietal pleura are freed from the immediately overlying tissues. Parenthetically, it should be said that we wait from two to three or four months following a manifestly unsuccessful thoracoplasty before we introduce the Lucite balls. For, by so doing, we are certain that the parietal pleura is sufficiently solid to minimize the danger of a pulmonary slough from ball pressure. We carefully avoid freeing the apex of the lung, for we want the pressure to be entirely lateral.

For the first three or four days following this procedure, but seldom longer than one week, the patient is often conscious of the clicking of balls in the chest when he moves around or coughs. After that time, I do not believe there has been a case where a patient has been conscious of ball movement.

On two occasions we have removed the balls several weeks after insertion. One because of pressure against a bronchus, which caused an almost constant harassing cough. This was relieved after some of the balls were removed. The other occasion was because of postoperative, nontuberculous infection. In both these cases, the sputum continued negative by culture.

We have had no deaths, and no complications that we have not been able to relieve. Our laboratory reports show 50 per cent negative sputum by culture in our series. We have used the Lucite pack only in cases where thoracoplasty has proved unsuccessful, and there seemed nothing else which offered a reasonably good chance for conversion of the sputum.

So I say, as I did in the beginning, that instead of being discouraged with the results we have obtained, we look upon the Lucite pack as a definite addition to our series of surgical procedures directed toward the cure of pulmonary tuberculosis. As in the choice of any operation for this disease, the important thing is the choice of the case selected for the particular operation contemplated.

DR. ROBERT KLOPSTOCK, Jamaica, N. Y.—I should like to make two technical remarks and one recommendation. If one has to resort to any extrapleural compression therapy using air or foreign body of a proved substance, this type of procedure should be carried out only if the pleural space has been tested for firm obliteration, prior to the operation. Extrapleural plombage is recommended as an operation of minimum trauma in cases with limited reserve. The perforation likely to occur into the pleural space if it has been free prior to operation may be a very grave complication, particularly in patients with limited reserve. The other technical point that I should like to make is that the extrapleural space should be absolutely dry, tested for being free of oozing before the foreign material

is placed into it. Exudate in the plombage bed can exert prohibitive pressure upon the mediastinum and by mobilizing the plombage itself creates "the floating" ineffective plombage.

The recommendation, which may sound dissenting, would be to regard the plombage as a temporary procedure. One should remove the plombage, irrespective of substance used (even if the result proved to be satisfactory) between six or eight weeks to six months after operation and to follow it up at the same time with a limited apical thoracoplasty. The plombage, a limited surgical procedure if performed with delicate care, served its purpose by creating a very selective collapse. In order to secure the permanency of this selective collapse it seems to be safer to replace the plombage with just a limited apical thoracoplasty than to rely on the plombage itself, for the foreign material stimulates the formation of a continuously shrinking fibrous capsule around itself. The slow shrinkage can exert a considerable pressure within the capsule. The least resistant point outside of the capsule is the surface of the damaged lung toward which the late perforations are likely to occur due to the protruding of the plombage substance if the plombage has not been removed in time. Late perforations may occur from six months up to two or more years following operation.

DR. TRENT.—I should like to say in closing that certainly we agree with Dr. Decker that the longer the cases are followed the poorer are the results. Our follow-ups, I should like to remind you, although not very long, have continued for a minimum of one and one-half years, the longest approximately three years. I have one slide I should like to show you, which illustrates one type of case included in the Duke Hospital series. Some of these cases like the one Dr. Brantigan illustrated, were selective, in that the area immediately over the cavity, which is approximately under the second rib in this case, were selected for collapse and the lung allowed to remain attached to the mediastinum. (Slides.)

This was done approximately two years before this report. The pictures show the patient approximately one year after operation and immediately after operation. When the patient was discharged the sputum was negative. While at home he resumed activity, had no symptoms, and was feeling well. We considered this a good result. A few months ago he began to develop symptoms and felt he should return for check-up. He returned two months ago and the film on the right was made. The smear was found loaded with acid-fast organisms two years after operation.

AN ANALYSIS OF VARIATIONS OF THE SEGMENTAL BRONCHI OF THE RIGHT LOWER LOBE OF FIFTY INJECTED LUNGS

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INTRODUCTION

NOTWITHSTANDING the general acceptance in this country of the Jackson-Huber terminology of the bronchopulmonary segments, and in the British Empire of the somewhat differently named but corresponding zones, there seems not to have been made, as yet, a statistical study of the variations of the lower lobe segments. Brock (1946) discussed these from the standpoint of the varying amount of territory a given segment can cover and published excellent figures of some of these injected specimens. However, there still remains the task of analyzing variations in the light of an empirically established prevailing pattern, that is, of determining the typical surface area of a segment and then noting how often, and by what other bronchi, this is supplied in whole or in part. Such an analysis would seem to be a necessary preliminary to accurate localization of lesions of the lung and to surgical resection of its segments.

The present study is restricted to the segments of the right lower lobe, but a companion article (by Berg, Boyden, and Smith), dealing with the segments of the left lower lobe, together with a summary of the differences between the two lobes, is being prepared for publication.†

MATERIAL AND METHODS

Fifty fresh specimens of right lower lobes have been used in this study. The technique of injection was that employed by Scannell (1947). After the hilar region had been freed of vessels and lymph nodes, the primary branches of the right lower lobe stem were dissected from the surrounding parenchyma for a distance of about 1.5 cm. In many instances the level of primary branching of each segmental bronchus was reached also. Following partial inflation with air to clear the passages, each segment was injected with hot colored gelatin at approximately 85° C. The colored gelatin was prepared by dissolving 75 Gm. of U.S.P. gelatin and 10 Gm. of potassium iodide in 500 c.c. of tap water at 100° C and adding 4 c.c. of appropriate vegetable dye to each 100 c.c. of gelatin. Simple aniline dyes available at the grocery store were used.

Immediately after injection of each segment, the lobe was plunged into cold tap water to aid in the gelling of the segment. After two minutes it was removed from the cold bath and another segment injected. Midway through the process the intersegmental margins, as revealed by the colored masses, were drawn on the surface with India ink. This step was repeated, following the completion of the injection process, but this time the surface of the lung was carefully dried with toweling, and under an electric fan, both before and after

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†For comparable analyses of the left and right upper lobes, see Boyden and Hartmann (1946), Scannell (1947); Boyden and Scannell (1948), and Scannell and Boyden (1948).

application of the ink. In this manner the intersegmental boundaries were preserved permanently.

After drawings were made of the anterior, posterior, and diaphragmatic views of each lung, the segmental bronchi and rami were dissected in order to check and correlate the pattern of the bronchial tree with that of the surface distribution of the injected gelatin. The fact that the lungs were solid masses of gelatin made this step much more difficult than would have been the case if one were dissecting lungs preserved by the usual embalming methods, but the essential points could be determined in each case. After the tree was in full view and drawn for the record, the stem was opened and the location of the bronchial orifices noted on the drawing.

The terminology employed in designating the bronchopulmonary segments is that proposed by Jackson and Huber (1943). The numerical designation of branches is that devised by Boyden (1945).

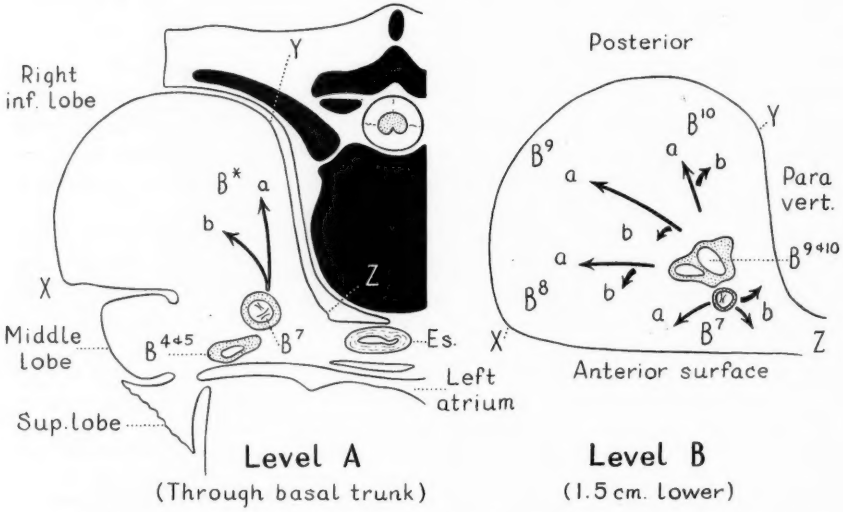


Fig. 1.—Transverse sections of right lower lobe at two levels: *Level A*, section just below orifice of superior segmental bronchus (B^6), to show origin of subsuperior bronchus (B^*) from common stem of $B^{8,\ 9+10}$; the branches of B^* are so lettered as to correspond to the overlying branches of B^6 (see Fig. 3). *Level B*, section just below origin of B^*, to show sectors of the lung supplied by the four basal bronchi (B^7, B^8, B^9, B^{10}). *X*, interlobar margin, separating anterior (interlobar) from costal surface of lobe; *Y*, costoparavertebral margin, separating paravertebral from costal surface of lobe; *Z*, pulmonary ligament, separating anterior from paravertebral surface. Arrows, at *Level B*, indicate direction of bronchi. The *b* branches of B^8, B^9, B^{10} are descending toward the diaphragm. Note that B^7a supplies the anterior surface, B^7b the paravertebral (and anterior) surface.

THE GROSS CONFIGURATION OF THE RIGHT LOWER LOBE

The right lower lobe somewhat resembles a cone that has been flattened anteriorly and medially, left rounded on its posterior and lateral side, and then telescoped from below. It thus presents four surfaces—a slightly curved anterior surface flattened by the upper and middle lobes, a paravertebral surface lying against the vertebral column and grooved by the esophagus, a costal surface molded by the curving ribs, and a concave diaphragmatic surface indented by the dome of the liver. As seen in cross section (Fig. 1) the anterior and costal surfaces are separated by the right-angled interlobar margin (X),

the costal and vertebral surfaces by an obtuse costoparavertebral margin (Y), and the anterior and paravertebral surfaces by the hilum and pulmonary ligament (Z).

The *costo-paravertebral margin* is barely noticeable in a fresh specimen, but it has been arbitrarily indicated by a line in the figures on the right side of Plates 1 to 4 (see Plate 1 for designation), since the position of the segments cannot be defined easily without such a landmark.

The *interlobar margin* is shown in the figures on the left side of Plate 1. This is the edge which marks the posterior border of the interlobar or oblique fissure. As pointed out by Brock (1942), this fissure begins at the fifth rib or the fifth interspace (one segment lower than is usually described in the anatomic texts) and follows the sixth rib down to the diaphragm.

The *anterior* (or interlobar) *surface* is not a flat plane but is often gently curved. Superiorly, as the lobe thins out and becomes nearly semicircular in cross section, the anterior surface faces somewhat laterally. This portion is usually fused in varying degree with the posterior segment (B^3) of the upper lobe. (See Plates 1 and 4, No. 40 and 49.) Inferiorly, the anterior surface faces more medially and is characterized by a fossa which we have termed the sulcus of the *inferior* vena cava (Plate 1) to distinguish it from the fossa for the superior vena cava which carries the anatomic term, sulcus venae cavae. The former is also a substantial groove. This was recognized by Braus (1924), who gave it the vernacular German name of *Delle*.

Laterally, the inferior portion of the anterior surface is crossed by the middle lobe. The lower border of the middle lobe usually passes from the hilum toward the lower lateral angle of this anterior surface. (See line separating subsegments B^8b and B^7a in specimen No. 39, Plate 4.) The position of this inferior margin of the middle lobe is not marked by any ridge or groove on the anterior surface of the lower lobe, nor have any of the fifty specimens presented a fusion of the middle lobe along this line. However, the proximal end of the middle lobe frequently fuses to the lower lobe at the inferior or lateral margins of the hilum (see No. 40, Plate 1).

The superior margin of the middle lobe coincides with the *interfissural crest* (Plate 1) which separates the surfaces on the inferior lobe that are covered, respectively, by the upper and the middle lobe. This line usually coincides with the division between upper and lower portions of the inferior lobe, the upper half representing the distribution of the superior segmental bronchus (B^6) and the lower half the zone supplied by the two basal segmental bronchi (B^{7+8}).

THE SUPERIOR SEGMENTAL BRONCHUS (B^6)

The superior segmental bronchus is the first posterior branch of the right lower lobe stem and is situated just inferior to the level of the orifice of the middle lobe bronchus and nearly opposite to it. It has three principal rami (Fig. 2, *A*): a medial (B^6a), a superior (B^6b) and a lateral (B^6c).* This does

*The medial is the paravertebral ramus of Brock (1943). It was originally designated the posterior ramus by Boyden (1945), but the additional information obtained in this study suggests that medial is more accurate than posterior. Also, in conformity to the method of numbering branches of the right lung in counterclockwise order, the letters designating these rami have been revised.

EXPLANATION OF PLATES

(Sketches rendered for publication by Lawrence B. Benson)

Sketches of injected fresh specimens of the right lower lobe of the lung, showing distribution of segmental bronchi. The large drawings on the left side display the anterior or interlobar surface; those on the right side, the costal surface as viewed posteriorly (X½). The smaller figures represent the diaphragmatic surface (X⅛).

Color scheme: *Yellow color,* segment supplied by superior bronchus (B^6); B^6a, b, c, distribution of its medial, superior, and lateral rami. *White color,* segment supplied by subsuperior bronchi (B^* and/or BX^*). *Blue color* (anterior surface), segments supplied by medial basal bronchus (B^7); a, b, distribution of anterior and medial rami. *Red color,* segment supplied by anterior basal bronchus (B^8); a, b, distribution of lateral and basal rami. *Blue color* (costal surface), segment supplied by lateral basal bronchus (B^9); a, b, distribution of lateral and basal rami. *Green color,* segment supplied by posterior basal bronchus (B^{10}); a, b, laterobasal and mediobasal rami. (The highest branch of B^{10} (BX^*) is left uncolored when it supplies the subsuperior zone.)

Plate 1.—*Specimen 43,* The superior segment (B^6) is typical as to size, angle of placement, and distribution of rami. The subsuperior segment (B^*) is typical in position (except that it is pushed somewhat laterally by the invading ramus of B^7) but it belongs in that small group (14 per cent) in which the segment is supplied only from level 2 of the stem bronchus, that is by a subsuperior proper (B^*). In this case, the bronchus arises just above the orifice of B9. The basal segments are typical except that the medial basal bronchus (B^7) is defective: although originating at its usual place, it consists of a medial ramus only (B^7b); the apparently missing anterior ramus (B^7a) is represented by an accessory branch (BX^7a) of the anterior basal, hence the latter (B^8) appears to extend atypically as far as the sulcus of the inferior vena cava.

Specimen 40, The superior segment (B^6) occupies the upper half of the lobe. Posteriorly, the lateral ramus (B^6c) predominates, and its displaced deep branch (‡) invades the subsuperior region (for normal deep branch, see Figs. 2*A* and 3). The subsuperior segment is thereby displaced somewhat laterally. It is supplied by the high dorsal branch (BX^*) of the posterior basal (B^{10}). The basal segments are atypical in that B^9 is absent as such, being represented by BX^9b, an accessory ramus of E^5b. Consequently in this specimen, the anterior basal segment extends more posteriorly than usual. B^7b fails to reach the diaphragm.

Plate 2.—*Specimen 48,* The superior segment (B^6) occupies an upper wedge-shaped zone of the lobe. Both it and the posterior basal segment (B^{10}) are shortened by the unusual paravertebral extent of the subsuperior segment. The latter is supplied by three bronchi—one subsuperior proper (B^*) which arises just beneath the orifice of B^7, and two accessory subsuperiors (BX^*) which arise several centimeters lower down from B^{10}a and B^{10}b. The lateral basal segment (B^9) is shortened by the unusual spread at B^8a.

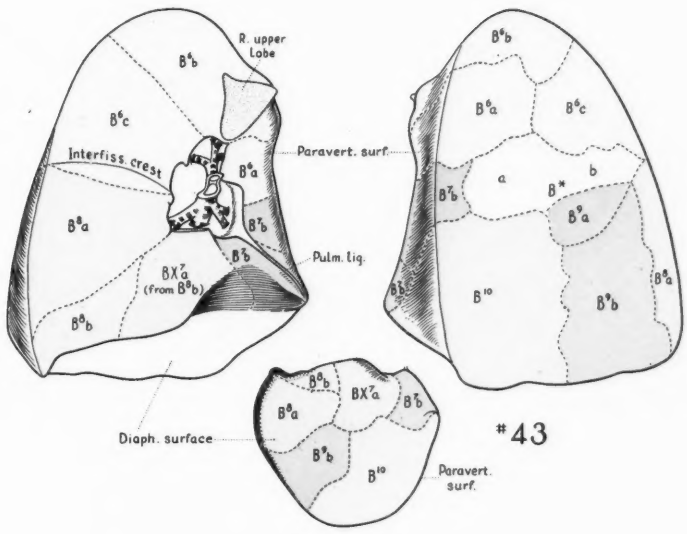
Specimen 42, The superior segment (B^6) occupies an oblique half or less of the lobe. Again ramus B^6c predominates posteriorly. The subsuperior segment is a small mid-posterior zone supplied by BX^* (10). The medial basal segment (B^7) is demarcated by a deep fissure which extends to the center of the diaphragmatic surface. The anterior basal (B^8) is anomalous in that while its bronchus arises from the usual position it consists of the lateral ramus only (B^8a). (This, in turn, has an accessory branch [BX^6c] which takes over part of the territory above the interfissural crest, that is, the zone usually supplied by the superior bronchus, B^6.) The basal portion of the anterior basal segment (BX^8b) is taken over by an accessory branch of the lateral basal bronchus (B^9).

Plate 3.—*Specimen 50,* The superior segment (B^6) is obliquely placed. In contrast to that of Specimen 40 (Plate 1) the segment in this specimen descends further anteriorly than posteriorly. Also B^6c predominates posteriorly. The subsuperior segment is supplied by two subsuperiors proper, the first B^* arising midway between the orifices of B^8 and B^9, the second B^* just above the orifice of B^9, yet, curiously enough, they terminate side by side. Because of the low origin of the second B^*, the accessory subsuperior (BX^*) was presumably forced to distribute much lower than usual; for this reason it has not been included in the subsuperior segment although its designation has been retained. This is also the only specimen encountered in which the lateral branch (B^9a) of the lateral basal segment reached the diaphragm. Otherwise, the basal segments are typical.

Specimen 46, This and No. 42 (Plate 2) illustrate that 38 per cent of specimens in which the subsuperior segment is supplied only by the BX^* ramus of B^{10}. It differs from that of No. 42 in that it extends into the paravertebral area. The other segments are typical.

Plate 4.—*Specimen 39,* This is the only specimen encountered in which there were two subsuperior bronchi arising exclusively from the stem of the posterior basal (B^{10}). Only the upper one, however, may properly be considered as supplying the subsuperior segment. Also, this specimen resembles No. 42 (Plate 2) in that B^8a takes over a portion of the superior segment by virture of its accessory ramus, BX^6c. It differs in that B^8b is normal in origin and not a branch of B^9 as in No. 42.

Specimen 49, This is the only specimen encountered in which the anterior basal segment (B^8) existed as a separate lobe. As shown in diaphragmatic view, the segment consisted of a cleft lobe which seemed to have protruded from its position between the medial basal (B^7) and lateral basal segment (B^9). This specimen is also the only one encountered which had a distinct cleft between superior and basal segments. It is also peculiar in the extent to which the superior segment is fused to the upper lobe.


R. upper Lobe
Interfiss. crest
Paravert. surf.
Pulm. liq.
Diaph. surface
Paravert. surf.
#43

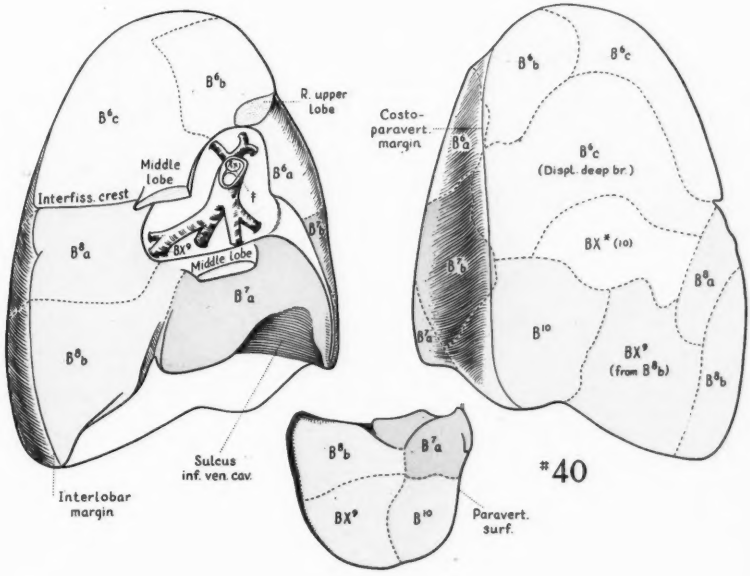
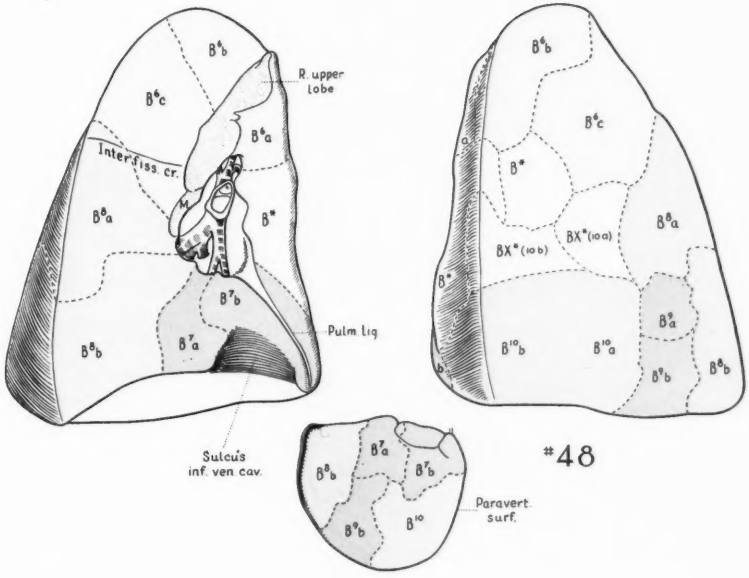

R. upper Lobe
Middle lobe
Interfiss. crest
Middle Lobe
Costo-paravert. margin
(Displ. deep br.)
Sulcus inf. ven. cav.
Interlobar margin
Paravert. surf.
#40

Plate 1.


R. upper lobe
Interfiss. cr.
Pulm lig
Sulcus inf. ven. cav.
Paravert. surf.
#48

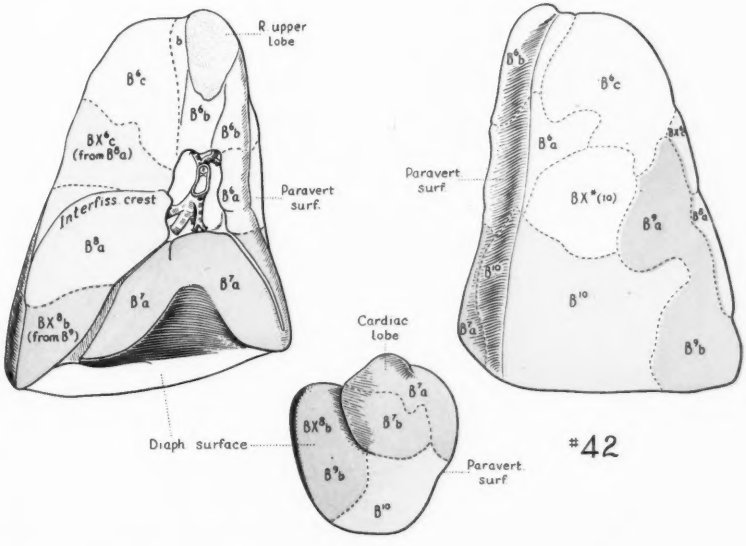
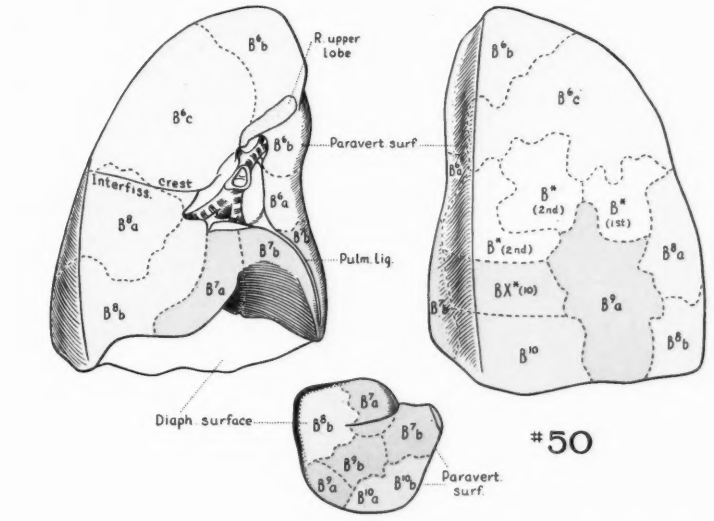

R. upper lobe
Paravert. surf.
Interfiss. crest
Paravert surf
Cardiac lobe
Diaph. surface
Paravert. surf.
#42

Plate 2.


R. upper Lobe
Paravert. surf.
Interfiss. crest
Pulm. lig.
Diaph. surface
Paravert. surf.
#50

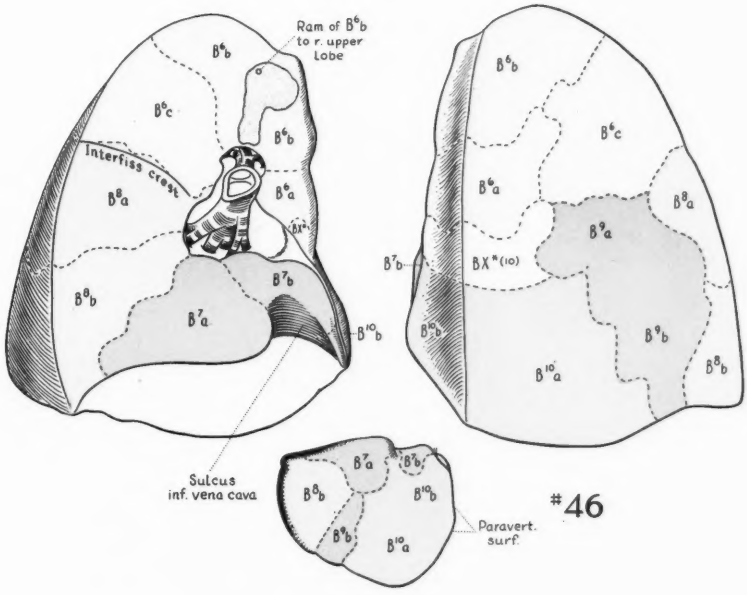

Ram of B6b to r. upper Lobe
Interfiss. crest
Sulcus inf. vena cava
Paravert. surf.
#46

Plate 3.

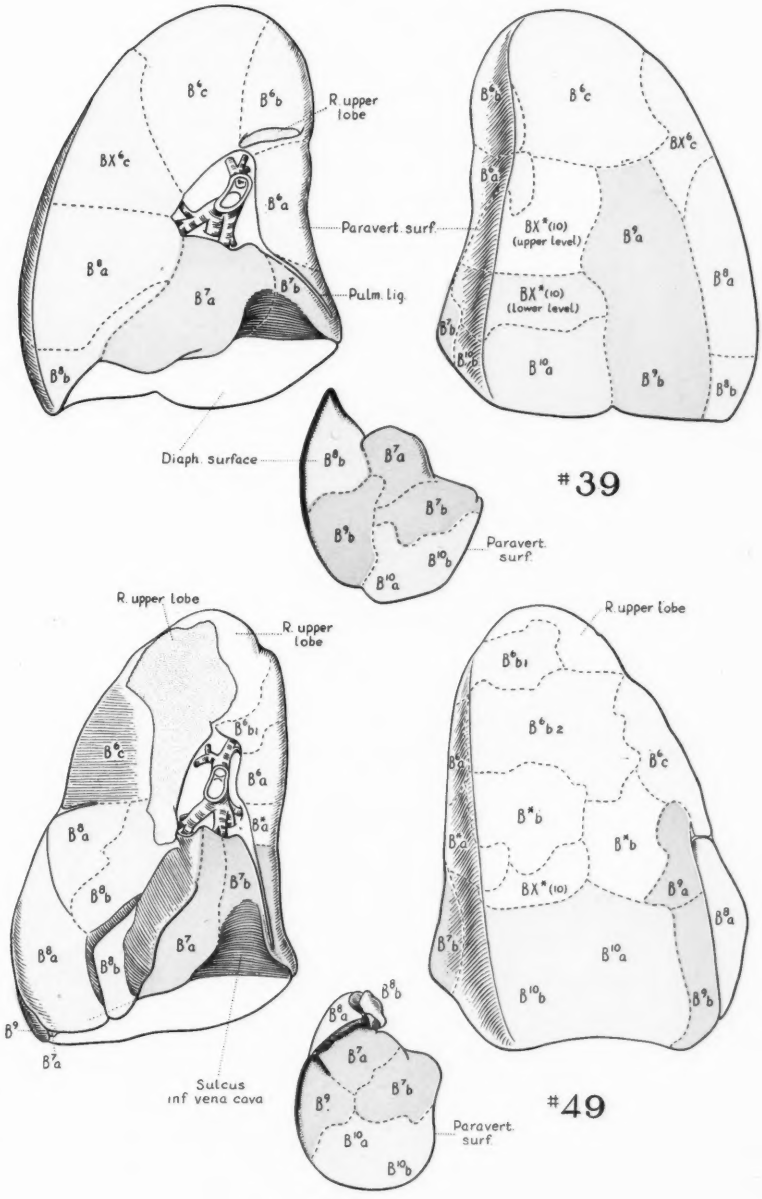

R upper lobe
Paravert. surf.
Pulm. lig.
Diaph. surface
Paravert. surf.
#39
R. upper lobe
R. upper lobe
R. upper lobe
Sulcus inf. vena cava
Paravert. surf.
#49

Plate 4.

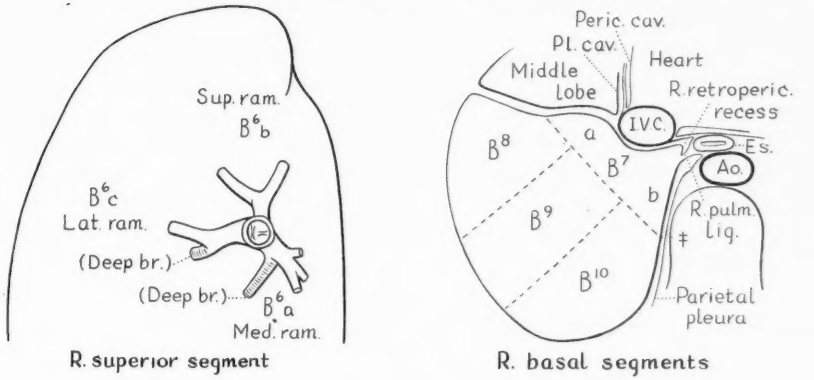


Fig. 2.—*A*, Anterior view of superior segmental bronchus (B^6), showing arrangement of its three rami—B^6a, B^6b, B^6c—in an embalmed specimen (see Fig. 7,*B*). While this represents the common type of bifurcation into B^6a + b and B^6c it is atypical in that it resembles a trifurcation. For more typical mode of branching, see B^6, Fig. 3, *C*.

B, Diagram of transverse section of the right lower lobe through the lower part of the basal segments. Note that the medial basal segment (B^7) is placed athwart the other basal segments (B^8, B^9, B^{10}) and that the latter exhibit a layering, B^8 overlapping B^9, and B^9 overlapping B^{10}. Also note that B^7 lies on both sides of the pulmonary ligament, B^7a typically extending as far as the lateral third of the anterior surface; B^7b reaching well down on the paravertebral surface (‡).

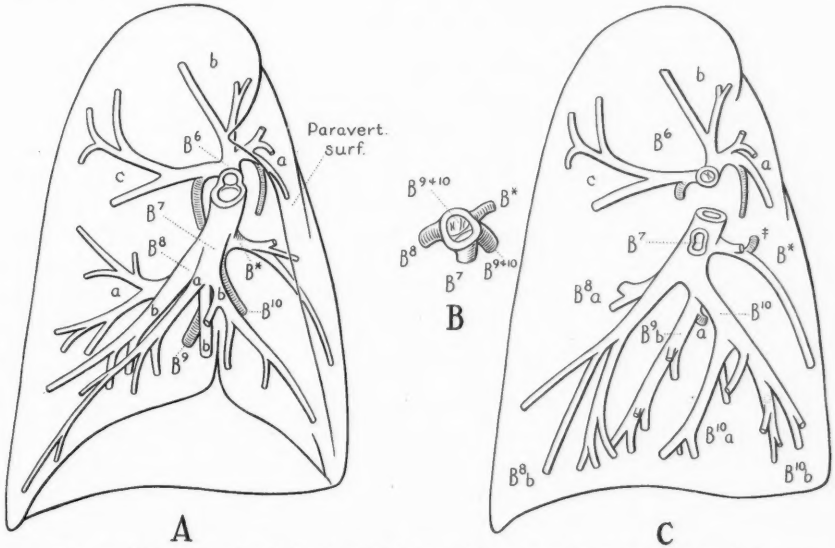


Fig. 3.—Dissection of embalmed right lower lobe as seen from anterior surface.

A, Superficial dissection of bronchi. This specimen is typical in respect to the level of origin of the segmental bronchi B*, arising from the common stem of B^8, B^9, and B^{10}, but below B^7. It is atypical in that the subsuperior bronchus (B*) is directed medially instead of posteriorly. For this reason, presumably, the medial ramus of the medial basal (B^7b) does not distribute to the paravertebral surface as it usually does. Correspondingly, the anterior ramus (B^7a) supplies the whole of the lower anterior surface, instead of the usual two-thirds of it. Finally, the deep branch of B^6c takes over part of the posterior distribution of B*.

B, Orifice of basal stem just below level of B^6.

C, Deeper view of dissection following removal of B^7, the medial basal bronchus. Note deep posterior branch of the subsuperior (‡) that was hidden in *A*. Note absence of BX* (10)—the high posterior branch of B^{10} which occurs in 86 per cent of specimens. B^6, superior segmental bronchus; B*, subsuperior bronchus; B^7, medial basal; B^8, anterior basal; B^9; lateral basal; B^{10}, posterior basal segmental bronchus.

not mean that B^6 is typically trifurcate, as implied by Brock (and suggested by the specimen drawn in Fig. 2): for the prevailing pattern (86 per cent) is a bifurcation, B^6c and $B^6a + b$; see Fig. 3 and Plate 2. (See also Table I, item 1.) The remaining items in the table demonstrate how rarely this pattern is modified. In only a few instances (10 per cent), is the segment supplied by an accessory branch of B^8 (see BX^6c, Plate 2, No. 42; Plate 4, No. 39), or by an accessory branch of the subsuperior bronchus, or directly by a branch from the basal stem (see footnotes, Table I).

TABLE I. THE SUPERIOR SEGMENTAL BRONCHUS (B^6)

1. *Bifurcates* into:			94%
1.* $B^6a + b$ and B^6c (Plate 2, No. 42)		86%	
2. Bizarre types		8%	
(1)	B^6c2 and $[B^6a + c1 + b]$	2%	
(2)	B^6c2 and $[(B^6a + b) + c1]$	2%	
(3)†	B^6a and B^6b	2%	
(4)‡	B^6b1 and B^6b2	2%	
II. *Trifurcates* into B^6a, B^6b, B^6c			6%

*In three specimens, part of B^6c arose from the basal stem (No. 17) or from B^8; see No. 42 (Plate 2) and No. 39 (Plate 4).
†B^6c arises as BX^6c from the basal stem.
‡B^6a arises as BX^6a from the subsuperior bronchus (B^8).
B^6c1 arises as BX^6c1 from B^8; B^6c2 from basal stem.

The amount of the lower lobe tissue that is aerated by B^6 is of considerable importance to the clinician since this is frequently involved in lung abscess and frequently separated from the basal segments. For these reasons it has seemed desirable to summarize the limits of the superior segments on all three surfaces of the lower lobe (Fig. 4). Row 1 shows the extent of this segment on the anterior surface, together with the percentages in which it occupies one-third, one-half, or two-thirds or more of the surface. Rows 2 and 3 supply similar data for the costal and paravertebral surfaces, respectively. However, these figures, suggestive as they are, do not in themselves yield the most significant information. This has been obtained by ascertaining the extent of this segment in each specimen and then finding into what groups these specimens can be arranged.

Two major patterns emerge from this analysis. In two-fifths of the specimens (38 per cent) the superior segment is obliquely fitted to the basal segments of the lower lobe. In the remainder (62 per cent), it caps the basal segments horizontally, according to conventional concepts. The whole group may be further subdivided according to whether the segment occupies the upper half

TABLE II. POSITION AND EXTENT OF THE SUPERIOR SEGMENT

Horizontal group		62%
Upper third	8%	
Upper half	52%	
Upper two-thirds	2%	
Oblique group		38%
Upper third	6%	
Upper half	26%	
Upper two-thirds	6%	

(78 per cent), the upper third (14 per cent), or two-thirds (8 per cent) of the lobe. Details are given in Table II.

In no instance was the superior segment cut off from the basal segments by a substantial incisure, as described by Dévé (1900) in 6 per cent of 180 infant lungs—the nearest to this condition being shown in Plate 4 (No. 49)—nor was it

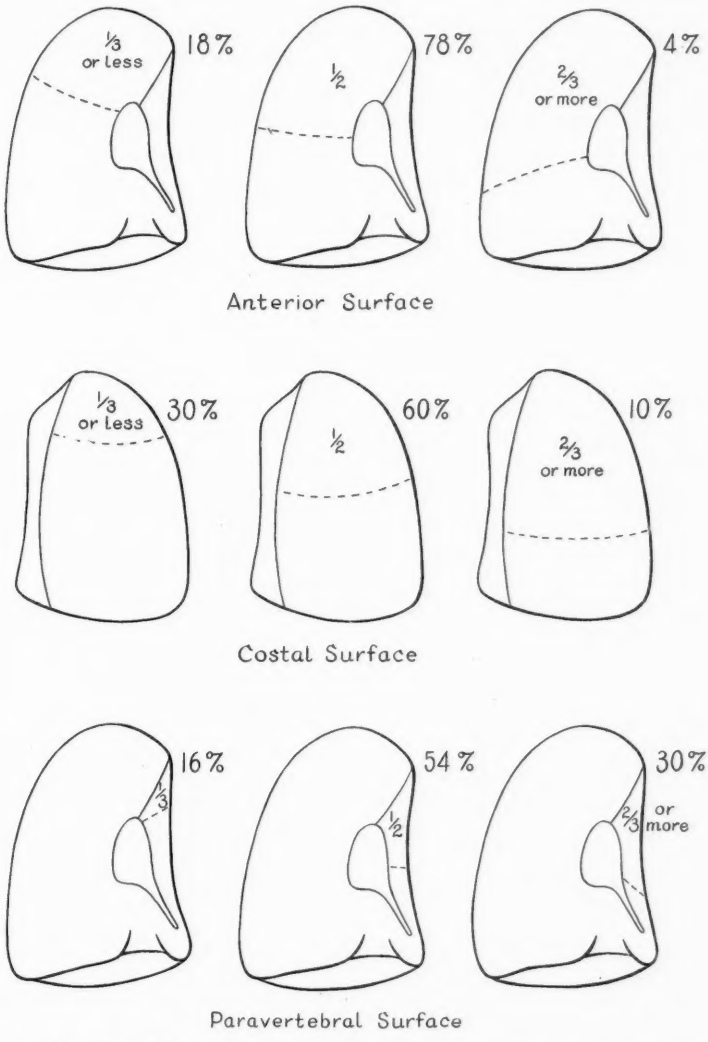


Fig. 4.—Diagrams illustrating varying extent of the superior segment on anterior, costal, and paravertebral surfaces. Percentages indicate frequency of given distributions.

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ever associated with the 36 per cent of incisures demarcating the medial basal segment in our series. Dévé observed a coexistence of these incisures in 11 per cent of infant lungs. (This figure includes incomplete fissures.)

The composition of the anterior and costal surfaces of the segment is displayed in Figs. 5 and 6, respectively. In the prevailing pattern (Fig. 5, 44 per cent), the anterior surface is supplied exclusively and equally by B^6b and B^6c. On the costal surface (Fig. 6), the two most common patterns are those in which rami b and c again divide the area (34 per cent), and those in which rami a, b, and c participate equally (30 per cent). On the paravertebral surface, which has not been plotted because of its constancy, B^6a is present and predominant in 98 per cent of cases. B^6b is the only other ramus which invades this area even to a minor degree and then only in 44 per cent of specimens.

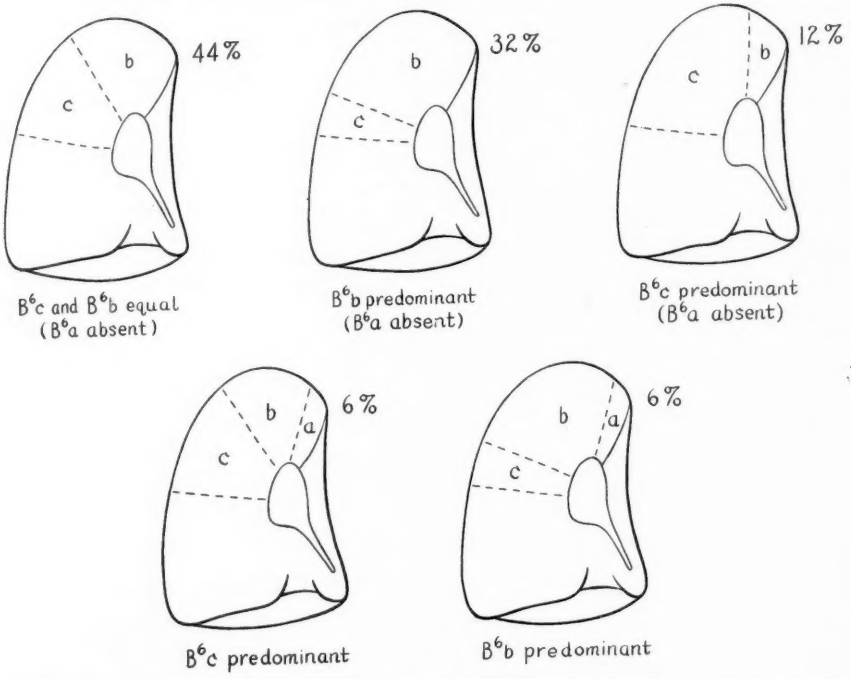

Fig. 5.—Diagrams illustrating varying extent of anterior surface of the superior segment supplied by individual rami of B^6.

Of the three rami just described, the *medial* (B^6a) is most worthy of attention since this is the one which descends furthest. This agrees with Brock's observation that the downward direction of this branch may be so increased that it runs almost vertically. Thus in 54 per cent of specimens (Fig. 4), it supplies one-half of the paravertebral surface (with or without a small portion of B^6b) and in another 30 per cent it covers two-thirds or more of the surface. Indeed, in two instances it almost reaches the diaphragm (see Fig. 9). From the stand-

point of resection of the segment, the importance of this cannot be overemphasized. Another point of interest is that in 38 per cent it develops a deep branch (Fig. 2, *A*) which takes over a substantial portion of the costal surface (see Plate 3, No. 46), but it was never found to predominate on that surface.

Next in interest is the *lateral ramus* (B^6c). In 84 per cent this has a deep posterior branch (Fig. 3, *A*) and occasionally (16 per cent) it may take over most of the costal surface of the segment (Plates 2 and 4, No. 42 and No. 39). In one instance, what is presumably to be interpreted as this deep branch arose coordinately with the common stem of $B^6a + b$ (see orifice ‡, Plate 1, left side, No. 40). Evidence for this may be found in Plate 3 (No. 50) where the typically placed deep branch of B^6c supplies a comparable zone.

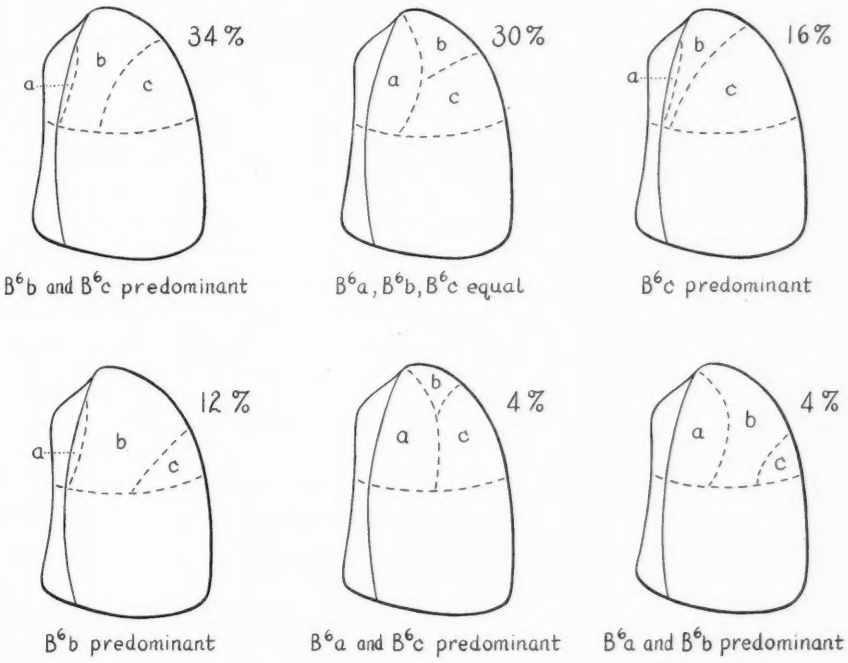

Fig. 6.—Diagrams illustrating varying extent of costal surface of the superior segment supplied by individual rami of B^6.

The *superior ramus* (B^6b) is distributed to both sides of the upper portion of the lobe.

From the standpoint of resection, the common stem of the three rami of B^6 is long enough to permit ligation in 92 per cent of specimens. The same is true of the common stem of $B^6a + b$ in 88 per cent and of the lateral ramus (B^6c) in 100 per cent of specimens.

In concluding discussion of the superior segment attention is again called to the fact that on the right side the upper lobe is usually fused to a greater or

lesser degree with the anterior surface of the superior segment where that plane curves to join the paravertebral surface. Usually this area is contiguous to B^6b. In this region (see Plate 3, No. 46), a branch of B^6b was observed to supply the parenchyma of the right upper lobe in five out of eleven consecutive specimens examined for this purpose. A similar situation was noted by Bell (1941). In a personal communication he stated that this fact was observed in a case of lobar pneumonia in which the consolidation extended from the right lower lobe to the adjoining area of the right upper lobe. Post-mortem dissection showed that the bronchial twig from the superior segmental bronchus supplied this area.

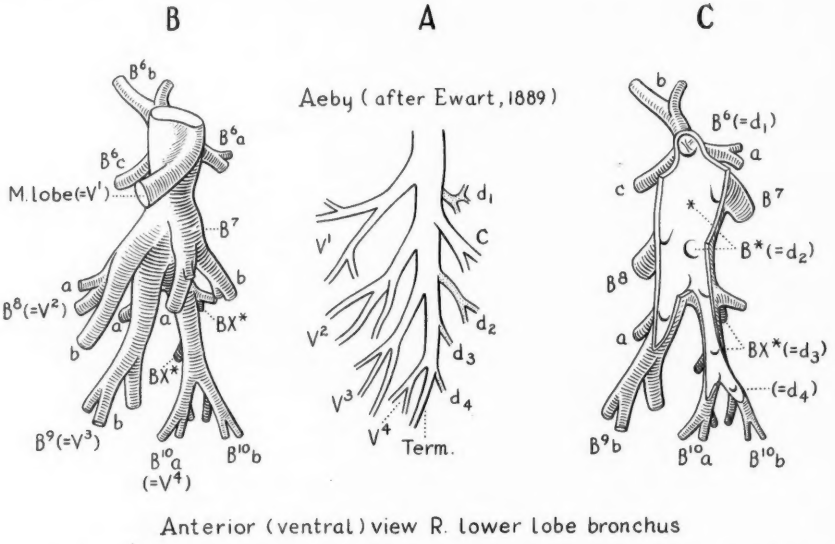

Fig. 7.—Sketches showing the origin and relations of posterior (dorsal) branches of the right lower lobe bronchus.

A, Aeby's diagram illustrating "monopodic" branching of a stem bronchus into a series of ventral (V^{1-4}) and dorsal (d_{1-4}) rami. *C*, cardiac bronchus, which does not fall into either of the two series. *Term.*, termination of "stem bronchus."

B, Dissection of embalmed specimen (No. 12, 816), for comparison with Aeby's diagram.

C, Same specimen as in *B*, with anterior wall of bronchial tree removed to show orifices of dorsal rami. In this particular specimen, five instead of Aeby's four dorsal rami occurred. (The first dorsal is represented by B^6.) A sixth is frequently found in certain other specimens at the point indicated by an asterisk, that is on the common stem of $B^{8, 9+10}$. Considering the six in order from above downward. No. 2 and 3 are considered as equivalent to Aeby's d_2. Dorsal bronchi on this part of the stem are here designated the subsuperior proper (B^*), No. 4 and 5 (on the stem of B^{10}) are considered as equivalent to Aeby's d_3 and are here designated as the accessory subsuperior, BX* (10). Bronchi arising in positions 2 to 5 usually supply the subsuperior segment, and not successively lower zones, as might be inferred from their position on the stem bronchus.

THE SUBSUPERIOR BRONCHUS (B^*)

The subsuperior bronchus, as indicated by its name, arises beneath the superior bronchus (B^6) either from the posterior portion of the basal trunk (B^*, Fig. 7, *C*) or from the upper portion of the posterior basal bronchus (BX*, Fig. 7, *C*). It is distributed to the middle level of the posterior costal surface of the lobe. It may extend onto the paravertebral or onto the lateral costal

surface. This is one of the zones which has been considered to be most vulnerable to aspirated material (Neil, Gilmour, and Gwynne, 1937).†

The first to recognize a series of dorsal or posterior branches on the stem bronchus of man was the Swiss comparative anatomist, Aeby (1880). He postulated a monopodic branching consisting of four ventral and four dorsal rami. In this he was guided by the prevailing mammalian pattern in which the thorax is long and narrow and the heart placed near the middle of the thorax. Such a scheme is shown at *A* in Fig. 7. Ewart (1889), however, took exception to this rationalization, stating that due to the different shape of the human thorax, which is compressed from front to back, with the heart placed inferiorly, the branching of the human bronchial tree is of necessity more radial, and more dichotomous, than monopodic (see *B*, Fig. 7). Furthermore it has been our experience that the four dorsal branches postulated by Aeby do not occur regularly in the right lower lobe bronchus of man, and that potentially there are six sites, as shown at *C* in Fig. 7: (1) the site of the first dorsal branch of Aeby (d_1), which is the superior segmental bronchus (B^6); (2) the dorsal branch between B^7 and B^8 (marked by an asterisk in *C*, Fig. 7); (3) the dorsal branch between B^8 and $B^{9, 10}$ (Aeby's d_2); (4) the dorsal branch at the upper end of B^{10}; (5) the dorsal branch at the middle of B^{10} (Aeby's d_3); and (6) the dorsal branch of $B^{10}b$ (Aeby's d_4). However, for the sake of simplification, dorsal branches 2 and 3 are here grouped together and considered as corresponding to Aeby's d_2, and dorsal branches 4 and 5 as corresponding to Aeby's d_3. It is thus convenient to refer to the subsuperior bronchus as arising either at level d_2 or level d_3, that is, from some portion of the main stem or from B^{10}, respectively. The first will be referred to as the *subsuperior proper*, with the designation B* (after Boyden, 1945) and the second as the *accessory subsuperior*, with the designation BX*.

In considering that both B* and BX* represent a subsuperior (in which view Brock also concurs) we have been guided by the fact that in the absence of a B* the BX* distributes to the subsuperior zone rather than to a distinctly lower level, a fact which again may be referred to the radial type of branching of the human lung.

Somewhat similar to Fig. 7, *C* is a sketch presented by Neil, Gilmour, and Gwynne (1939), showing the opened bronchial tree of a 4-year-old child in which three subsuperiors are present (their Fig. B). The first two correspond to the d_2 level, and the third—if the specimen had had the usual high bifurcation of B^{9+10}—to the d_3 level, as postulated in Fig. 7, *C*. In general, these authors

†At first unaware of Ewart's earlier identification of it (as the "lesser posterior horizontal") they claimed discovery of a new segment—naming it the "subapical"; and, indeed, it had been overlooked by Kramer and Glass (1932). Neil's excellent description of it, with emphasis on its clinical importance is as follows:

> "The posterior region of the lower lobe is supplied, according to all previous publications to which we have had access, by the apical bronchus [B^6] in the upper portion and the paravertebral [B^{10}] in the lower. We are convinced that an intermediate separate segment exists and can be demonstrated, and that it is the site of localized pathological change, as shown by radiology and bronchoscopy. It is an anatomical gap in the distribution between the apical and paravertebral bronchi in all illustrations we have seen. We have found that it is supplied by these subapical bronchi [B* and BX*]. This has been shown by inflation in specimens and by lipiodol fillings in patients."

stated that there are one to three present, and this corresponds to our observations; for inspection of Table III (Item I) will show that one or more subsuperiors are present in every specimen. In 50 per cent only one occurs (Item II:1); in 34 per cent two are present (Item II:2); in 14 per cent three occur (Item II:3), and in one specimen even four such dorsal rami were noted (Item II:4).†

TABLE III. SUBSUPERIOR BRONCHI (B* AND BX*)

I. Frequency of occurrence in 50 specimens			
1. Arises exclusively from level d_2 (as branch of main lower lobe bronchus between the superior and the basal segmental bronchi)			14%
2. Arises exclusively from level d_3 as an accessory subsuperior, BX*(10), i.e., one or more from the posterior basal bronchus (B^{10})			38%
3. Arises from both levels in same specimen			48%
			100%
II. Detailed analysis			
1. Subsuperior represented by single bronchus			50%
(1) Arising at d_2 level (as B* proper)		14%	
(a) From common stem of $B^{8, 9, 10}$	6%		
(b) From common stem of $B^{9, 10}$	8%		
(2) Arising at d_3 level, as BX*(10)		36%	
2. Subsuperior represented by two bronchi			34%
(1) One from level d_2 (B*), and one from level d_3, as BX*(10)		32%	
(2) Two from level d_3, as BX*(10)		2%	
3. Subsuperior represented by three bronchi			14%
(1) Two from level d_2 (B*) and one from level d_3, as BX*(10)		10%	
(2) Two from level d_3, as BX*(10) and one from level d_2, as B*		4%	
4. Subsuperior represented by four bronchi (Two from each level)			2%
			100%
III. Origin of B* (at d_2 level). (Items I:1 and I:3)			62%
1. From basal stem (level of B^7)		12%	
2. From common trunk of $B^{8, 9, 10}$		22%	
3. From common trunk of $B^{9, 10}$		14%	
4. Two: one from common trunk of $B^{8, 9, 10}$ and one from $B^{9, 10}$		6%	
5. Two from common trunk of $B^{9, 10}$		8%	

At this point one may ask why Neil, Gilmour, and Gwynne have not been followed in giving the subsuperior the status of a segmental bronchus. The answer lies in their observations and ours, namely, that the number and level of origin of bronchi supplying this middle posterior zone are variable, hence it is less confusing to give it a designation (B*) that is noncommittal as to numerical sequence.

Another point made by Neil and his co-workers is that they have observed the subsuperior (subapical) "arising from the dorsal, mesial, or lateral aspect of the right main bronchus." Similarly, we have found that occasionally B* inclines to the medial or lateral side. In the former position it may give rise to the medial ramus of the medial basal bronchus (B^7b)‡; but in general

†It should be pointed out that the precise level of branching of the bronchi can be determined only by opening the stem bronchus. That is, one cannot always tell from the external view whether a dorsal ramus is originating from the upper part of B^{10} or the lower portion of B^{9+10}. This emphasizes the necessity of preoperative bronchoscopy or bronchography.

‡In Fig. 3A, B* is medial and is taking over part of the territory of B^7, but has not replaced it.

Brock's description applies to the usual arrangement, namely that "typically [the subapical] arises from the posterior wall of the lower lobe bronchus—as it were, in series with [the apical bronchus]—. When it does not arise from the lower lobe stem it may originate from the posterior basal bronchus—still roughly parallel to [the apical bronchus]." Using Brock's definition, however, we have found the subsuperior occurring in 100 per cent of our smaller series of dissected specimens, in contrast to the 48 per cent incidence in his larger series of 170 bronchograms.

Hitherto the subsuperior has been discussed from the standpoint of its origin on the bronchial tree. Equally important is its distribution on the surface. Turning first to the subsuperior proper (B*), analysis of the fifty injected specimens shows that in all but one of the thirty-one specimens in which it occurs (Table IV), it aerates at least the posterior sector of the right lower lobe. In 36 per cent it distributes to at least the posterior and the adjacent paravertebral area, and in 32 per cent it aerates at the least the posterior and the adjacent posterolateral area. It would seem, therefore, that to be a subsuperior proper this bronchus must supply at least the posterior sector but that it may spread either medially (B*, Plate 2, No. 48) or laterally (B*, Plate 1, No. 43) or in both directions (B*, Plate 4, No. 49).

For percentages of types see Table IV.

TABLE IV. DISTRIBUTION OF THE SUBSUPERIOR (B*)

AREA	PER CENT
Posterior and adjacent paravertebral	20
Posterior and adjacent posterolateral	16
Posterior, paravertebral, and posterolateral	16
Posterior alone	8
Posterolateral alone	2

When the subsuperior proper is absent, its zone is usually supplied by a dorsal ramus arising from the posterior basal bronchus (B^{10})—level d_3 of Aeby. This is the ramus we have called the accessory subsuperior, BX* (10). It, too, almost always supplies at least the posterior sector (Plate 2, No. 42) but may spread medially to the paravertebral area (Plate 3, No. 46) or laterally to the posterolateral zone; or it may be displaced medially or laterally (Plate 1, No. 40) by one of the deep branches of B^6. For percentage of types, see Table V.

TABLE V. DISTRIBUTION OF THE ACCESSORY SUBSUPERIOR (BX*) IN THE ABSENCE OF A SUBSUPERIOR (B*)

AREA	PER CENT
Posterior alone	28
Posterior and adjacent paravertebral	4
Posterior and adjacent posterolateral	4
Posterolateral alone	2
Total	38

When both the subsuperior proper and the accessory subsuperior are present (48 per cent), the latter either distributes to the area beneath the former (BX*, Fig. 9) or shifts medially, or to the lateral side of it (BX*, Plate 2, No. 48), or telescopes it (Fig. 8). In only one-sixth of these specimens does BX*(10)

supply so low a zone as to be considered part of the posterior basal rather than the subsuperior segment (namely, No. 50, Plate 3).†

When there are two accessory subsuperiors (8 per cent), one may lie below the other, in the posterior sector, or they may lie side by side (BX*, Plate 2, No. 48). If the lower one distributes too inferiorly it may be considered a part of the posterior basal segment (namely, No. 39, Plate 4).†

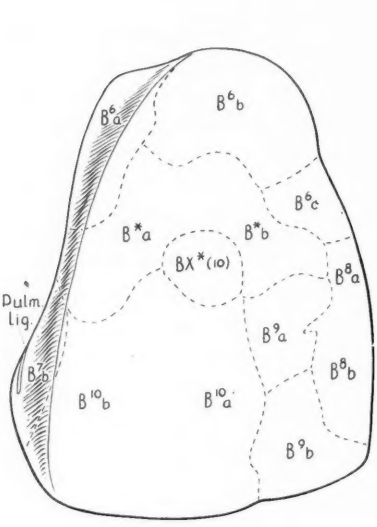

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Fig. 8.

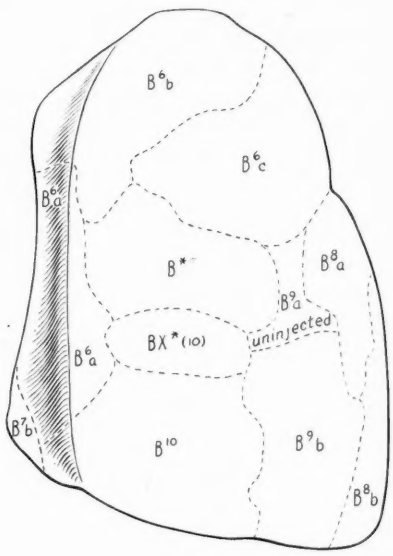

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Fig. 9.

Figs. 8 and 9.—The costal surface of two injected specimens showing varying distributions of the subsuperior proper (B*) and of the accessory subsuperior bronchus (BX*). In each specimen B* arises at a level between B^7 and B^8, and BX* several centimeters below this from B^{10}, but in No. 16, BX* telescopes the zone supplied, by B*, while in No. 38, it lies inferior to it. These specimens belong to that 48 per cent of lungs in which the subsuperior proper and the accessory subsuperior are present in the same lobe.

Specimen No. 38 also illustrates the fact that B^6a, the medial ramus of the superior segment, may take over almost the whole of the paravertebral area. In other words the superior segment may extend nearly to the diaphragmatic surface.

Of special interest are the 12 per cent of specimens in which two subsuperiors are present in addition to one, or even two, accessory subsuperiors (Table III, Item II:3[1] and II:4). In the former type, one B* may arise above the other but they distribute to zones lying side by side, as B* (first) and B* (second) in Plate 3, No. 50. Thus, they simulate the zone supplied by the two branches of a single subsuperior (B,* a and b, Plate 1, No. 43; see also Fig. 1,*A*).

†In such specimens the bronchi which by origin were destined to be accessory subsuperiors have been deflected downward by other bronchi. In these few specimens one thus encounters a transitional pattern of Nature which confounds man-made classifications. We have chosen to meet the dilemma by retaining the number BX*, to indicate that the bronchus has the same origin as that which supplies the subsuperior zone, but giving the zone the color of the posterior basal segment (see Plate 3, No. 50).

THE BASAL BRONCHI (B^{7-10})

The basal bronchi are four in number and arise from the basal trunk—that portion of the lower lobe bronchus which is distal to the superior segmental bronchus (B^6). As seen in cross section (Fig. 1,*B*), the major branches of the basal stem are seen to radiate in different directions. The *medial basal* (B^7), the highest branch, arises anteriorly (Fig. 3,*A*) and is distributed to the anterior and paravertebral surfaces (Fig. 1). The next branch, the *anterior basal* (B^8), arises anterolaterally (Fig. 3,*C*) and distributes to the anterior surface and lateral costal area. The next two segmental bronchi arise from a common stem which passes downward in a somewhat posteromedial direction (Fig. 3,*C*). The *posterior basal* (B^{10}) continues the direction of the common stem and distributes to the paravertebral surface and posterior costal surface. The *lateral basal* (B^9) diverges in a posterolateral direction, supplying the posterolateral sector of the costal surface. For this reason it appears to be a lateral branch (V_3) of the stem bronchus of Aeby (Fig. 7,*A*). It is useful to think of it this way provided one does not accept all the implications of the Aeby theory.

Another useful concept is the one advanced by the Russian surgeon Melnikoff (1922)—namely, that in dissecting the lung the lower lobes seem to separate into a series of oblique overlapping layers. We would restrict this to the last three basal zones (B^8, B^9, B^{10}) and add that B^7 is applied at right angles to these (Fig. 2,*B*; compare also with overlapping bronchi in Fig. 7,*B*).

1. *The Medial Basal Segmental Bronchus* (*B^7*).—The medial basal segmental represents the cardiac or infracardiac bronchus of mammals, which in most species occupies the space between the heart and the diaphragm. Its position on the stem is also characteristic, it being the only basal bronchus which constantly arises anteromedially (see Fig. 7). Because of this position one would anticipate that it would distribute to both the anterior and the paravertebral surfaces. Such is the case in 78 per cent of the 50 specimens examined.† Typically, it divides into an anterior (B^7a) and a medial ramus (B^7b), the line between the two falling, usually, in the sulcus of the inferior vena cava (Plate 4, No. 39).

In 14 per cent, B^7 was found to be absent as such—that is, no bronchus having that origin was distributed to that territory. In such specimens the anterior and paravertebral components of the zone were aerated by two displaced bronchi, one being an accessory branch of the anterior basal (BX^7a from B^8b) and the other an accessory branch of the subsuperior (BX^7b from B^*). In the remaining specimens (8 per cent), B^7 supplied only the anterior surface but these were peculiar in that while B^7 arose normally in one specimen (namely, Plate 3, No. 46), both of its rami were distributed to the anterior surface exclusively (see also Fig. 3,*A*). In the other three, only the anterior ramus arose in the usual position, the medial ramus originating as an accessory bronchus of the subsuperior (B^*) or of the posterior basal (B^{10}).‡

†These observations are thus in accordance with the recent figures of Lucien and Beau (Mattei, 1946) but at variance with Jackson and Huber's account of the extent of this segment. Their figures show no distribution of the medial basal bronchus on the paravertebral surface.

‡It is interesting to note that Huntington (1920) used the cardiac bronchus of mammals as an example of the method of formation of accessory bronchi. He said, "It happens occasionally that a peripheral lung segment receives its bronchial supply from two sources. The terminal alveoli of each contributing bronchus are closed off within the segment of the district which it supplies and do not communicate with those of its colleague. Together the two components make up a topographical pulmonary district ordinarily served by a single bronchus."

TABLE VI. MEDIAL BASAL SEGMENTAL BRONCHUS (B^7)

I. B^7 present on both sides of the pulmonary ligament			78%
1. Distributes to more than one-half of the lower anterior surface		54%	
2. Distributes to one-half or less than one-half of the lower anterior surface		24%†	
II. B^7 wholly anterior			8%
1. Both rami present (No. 46, Plate 3; Fig. 3)		2%	
2. B^7 present but one ramus displaced		6%	
(1) Medial ramus (BX^7b) from B^*	4%		
(2) Medial ramus (BX^7b) from B^{10}	2%		
III. B^7 absent as such			14%
BX^7a from B^8		14%	
BX^7b from B^*	12%		
from B^{10}	2%		

†In one specimen (No. 43, plate 1) B^7 consisted only of a medial ramus (B^7b). BX^7a arose from B^8 .

The extent of the anterior surface supplied by B^7, as measured along the diaphragmatic margin, is variable. According to Brock, "it commonly occupies less than a half of the anterior margin," but as may be seen in Table VI (Item I:1) it occupies more than one-half of the anterior surface in two-thirds of the typical specimens. If all are included that have a B^7 of typical origin (86 per cent), B^7 occupies two-thirds or more of the anterior surface in 50 per cent of specimens, one-half in 28 per cent, and only one-third or less in 8 per cent.

Its paravertebral extent is also variable, ranging from an area exceeding one-half of the vertical extent of that surface (Plate 1, No. 40) to a small zone at the extreme lower end of the lobe (Plate 2, No. 48). In only one instance did it invade the costal surface (Plate 1, No. 43). When it fails to occupy the paravertebral area or supplies only a minimal portion of it, the remainder of its territory is taken over by adjacent bronchi—B^6a (Fig. 9), or B^* (Plate 2, No. 48), or a medial branch of B^{10} (Plate 3, No. 46), or any combination of these.

Of special interest are the cases in which B^7 is absent (Item III, Table VI), or wholly anterior (Item II), with the medial ramus (B^7b) developing as an accessory branch of the subsuperior. In such instances the subsuperior arises from a posteromedial position (see B^*, Fig. 3, *A*), at the same level on the tree as B^7. In this position it could well be mistaken for the latter, and apparently that is the interpretation given by Nelson in Fig. 2 of his 1934 article. This led him to minimize the importance of the medial basal segment: "On the right side there is a separate cardiac branch arising from the medial wall of the lower lobe bronchus; it runs downward and slightly inwards and backwards, to supply a small area just below the root of the lung. On account of its small size, this bronchus will not be considered again."

Dévé (1900) noted an incisure on the diaphragmatic surface in 35 per cent of 180 lungs. From its position he judged that this incisure demarcated a zone corresponding to the cardiac lobe of mammals. A similar incisure of varying depth was observed in 38 per cent of the 50 specimens injected in this laboratory. In all but 2 per cent, the incisure marked the lateral boundary of the medial basal segment. Two-thirds of these were pronounced and some would have been demonstrable roentgenologically. All of them would have been noted upon surgical exploration.

In this connection, a word must be said about the relation of the pulmonary ligament to the medial basal segment. Both Dévé's Fig. 6 and Brock's Fig. 94 portray the pulmonary ligament as reflected from the lung at the posterior boundary of B^7 (see ‡, Fig. 2, *B*). In the large majority of specimens, however, the pulmonary ligament is reflected from the lung just upon or just posterior to what these authors call the "horn-like projection of the cardiac segment." In other words the right "retro-pericardiac recess" is shallow, necessitating severance of the pulmonary ligament in an interlobar approach for resection of the medial basal segment.

Because of the fissures just noted, and the superficial position of the segment, and the accessible (high) origin of the bronchus, it is felt that this zone constitutes a surgical unit which may be readily resected. Indeed, there is at least one published instance (Scannell) in which this has been successfully accomplished, for removal of a bronchiectatic segment.

2. *The Anterior Basal Segmental Bronchus (B^8).*—The anterior basal segmental bronchus is the second of the basal bronchi and arises a little below B^7 from the anterolateral aspect of the basal trunk (Figs. 3, *B* and 7). It is remarkably constant in its distribution, dividing into lateral (B^8a) and basal (B^8b) rami.

The former supplies the lower middle-anterior and lateral costal surfaces of the lobe (Plate 3, No. 46). In only 4 per cent of the fifty specimens could it be traced to the diaphragm. The upper limit of its distribution coincides with the *interfissural crest*, a more or less inconspicuous ridge on the lower lobe marking the junction of the upper and middle lobes. In 4 per cent of specimens it gives off an accessory branch (BX^6c) which extends above the interfissural crest to invade the territory of the superior segment (Plate 2, No. 42; Plate 4, No. 39).

The basal ramus (B^8b) distributes to the lower anterior (interlobar) and lower lateral costal surfaces of the lobe in addition to the anterolateral diaphragmatic surface (Plate 2, No. 48). Its extent on the interlobar surface is variable. Usually, it occupies only the lateral third (50 per cent of specimens), as measured along the inferior margin. In 16 per cent it extends medially into the sulcus of the inferior vena cava by virtue of an accessory ramus, BX^7a (Plate 1, No. 43). In one instance B^8b was displaced, occurring as an accessory ramus of B^9 (Plate 2, No. 42). In still another specimen the anterior segment occurred as a bifid lobe (Plate 4, No. 49). This was the only example of an incisure between B^8 and B^9.

In three of the four specimens in which B^9 was absent, B^8b spreads more posteriorly; in the fourth it has an accessory bronchus (BX^9b; Plate 1, No. 40).

In concluding this section, we believe that the anterior basal segment, like the medial basal, is readily resectable; for its bronchus has a high anterior origin, its distribution is fairly constant, and its medial border is frequently marked by an incisure (36 per cent of specimens).

3. *The Lateral Basal Segmental Bronchus (B^9).*—The lateral basal segmental bronchus usually arises from the anterolateral aspect of the short common stem of B^9 and ^{10} (Fig. 7, *B*). In only 10 per cent of specimens was this a long com-

mon stem. In another 8 per cent, B^9 was entirely absent as such. We agree with Brock that it is smaller than the anterior basal (B^8) and appears much more like a secondary branch of the posterior basal (B^{10}) than an important independent branch. It is distributed to the posterolateral sector of the basal half of the lower lobe (Plate 2, No. 48).

Almost immediately after its origin the bronchus divides into lateral (B^9a) and basal (B^9b) rami. The lateral branch, when present (86 per cent), is quite constant both in origin and in distribution, supplying in 74 per cent of specimens the posterolateral area adjacent either to the subsuperior bronchus (Plate 4, No. 49) or to the displaced subsuperior bronchus (Plate 3, No. 46). In a fifth of this 74 per cent, the lateral ramus even encroaches upon the subsuperior zone (see Plate 3) and might be considered to contribute to that interpolated segment. An example of its infrequent lower position may be seen in Plate 2 (No. 48) and Plate 3 (No. 50). The latter is the only case in which it extended to the diaphragm. In the 14 per cent of specimens in which it is absent its place is taken by a subsuperior (10 per cent; Plate 1, No. 40) or by the lateral ramus (B^8a) of the anterior basal (4 per cent).

The basal branch (B^9b) supplies the inferior posterolateral costal area and the corresponding sector of the diaphragmatic surface (Plate 4, No. 39). In the 8 per cent of specimens in which it was absent its place was taken by B^8b—by spread of that ramus in three instances, and by an accessory branch of that ramus (BX^9b) in one instance (Plate 1, No. 40).

4. *The Posterior Basal Segmental Bronchus* (*B^{10}*).—The posterior basal segmental bronchus appears to be the continuation of the right lower lobe bronchus, particularly the branch here designated as $B^{10}b$ (Figs. 3 and 7) which extends to the inferior posteromedial corner of the lobe. In dissecting specimens it has been found most useful to locate this ramus first, then the other branches may be quickly identified.

Typically B^{10} breaks up into three major branches. The highest (formerly numbered $B^{10}a$, Boyden 1945) has already been demonstrated to be in reality a displaced subsuperior, and accordingly has been redesignated $BX^*(10)$. It will be recalled that this is a fairly constant branch occurring in 86 per cent of specimens (see Table III).

Having disposed of this high dorsal branch (from Aeby's level 3), let us now consider the two major branches of the posterior basal, herewith designated the laterobasal ($B^{10}a$) and mediobasal ramus ($B^{10}b$).† In over one-half the specimens (58 per cent), $B^{10}b$ arises somewhat behind $B^{10}a$, but in virtually all cases both rami are distributed to both the costal and diaphragmatic surfaces. In addition, $B^{10}b$ is distributed to the paravertebral surface (for example, Plate 4, No. 49). Occasionally (10 per cent), a particular paravertebral branch of B^{10} may be noted, but it is not constant enough to merit a name.

SUMMARY

The distribution and arrangement of the segmental bronchi of the right lower lobe have been studied in fifty fresh lungs by the following methods: (1) the successive injection of the principal bronchi by differently colored

†These are the rami formerly designated by Boyden (1945) as $B^{10}b$ and $B^{10}c$.

gelatin masses; (2) the accurate sketching of each injected lobe, to record the surface distribution of the bronchi on interlobar, costal, and diaphragmatic surfaces; (3) the dissection of each lobe, to verify the distribution of bronchi, and to ascertain their mode of branching.

From these data it has been possible to establish the prevailing pattern empirically, and to analyze the principal variations in the light of that knowledge.

As viewed from the interlobar surface the right lower lobe may be divided into superior and basal zones, the line of separation usually falling at the interfissural crest. Posteriorly, an interpolated, "subsuperior" zone can be demonstrated between superior and basal segments in 100 per cent of specimens (vide infra).

The Superior Segment.—Since the superior segment is resectable, it is important to know its extent. Prevailingly, it "caps" the basal segments horizontally, but in 38 per cent of specimens it is obliquely fitted to the basal segments. Usually (in 78 per cent), it occupies the upper half of the lobe, but it may occupy two-thirds of the lobe (8 per cent) or only one-third (14 per cent). On the paravertebral surface it may extend two-thirds (or more) of the way to the diaphragm (30 per cent of specimens).

The superior segmental bronchus (B^6) has three constant rami—a medial (B^6a), a superior (B^6b), and a lateral ramus (B^6c)—but it rarely trifurcates (6 per cent), the prevailing pattern (86 per cent) being a bifurcation into $B^6a + b$ and B^6c. On the interlobar surface the superior segment is usually fused to a greater or lesser degree with the right upper lobe; and in five out of eleven consecutive specimens examined for this purpose a branch of B^6b was observed to supply the adjacent portion of the right upper lobe. This may explain how consolidation of the lower lobe, in lobar pneumonia, may extend into the adjoining area of the upper lobe.

The *subsuperior* zone is a constant posterior zone interpolated between superior and basal segments. In this series of dissections it has been identified in 100 per cent of specimens, as contrasted with Brock's identification of only 48 per cent in a series of bronchograms. It is supplied by one or more posterior bronchi of inconstant origin. These arise from levels 1.5 to 5 cm. below the orifice of the superior bronchus (B^6). For this reason, the bronchi supplying this zone have been indicated by the noncommittal designation, "B*" (Boyden, 1945).

In general the principal levels of origin correspond to Aeby's dorsal bronchi, d_2 and d_3 (the first dorsal being the superior bronchus, B^6). That posterior bronchus which arises from Level 2 (that is, from the common stem of $B^{8, 9, \text{ and } 10}$ or B^{9+10}) is designated the "subsuperior proper," B*. That posterior bronchus which arises from Level 3 (that is, from the trunk of B^{10}) is designated the "accessory subsuperior," BX*(10).

In about one-half the specimens (48 per cent), the subsuperior bronchi arise from both levels, and range in number from two to four. In the remaining

one-half (52 per cent), there is only one subsuperior bronchus—more commonly BX* (38 per cent), as against 14 per cent with a subsuperior proper.

Primarily the subsuperior zone lies in the posterior sector of the lobe, but it may spread laterally or medially. It is a common site of lung abscess.

The *basal bronchi* are four in number and tend to radiate from above downward into the anteromedial, anterolateral, posterolateral, and posteromedial sectors of the lobe, respectively.

The *medial-basal segmental bronchus* (B^7) is the highest in origin. It distributes to the zone that lies on either side of the pulmonary ligament (78 per cent of specimens), and is placed athwart the other basal zones—that is, it occupies a plane virtually at right angles to the others. This segment corresponds to the cardiac lobe of mammals. Therefore, it is not surprising to find an incisure of varying depth separating it from the anterior-basal segment in 36 per cent of specimens. In two-thirds of this number the incisure is possibly deep enough to have been visible in a roentgenogram.

The bronchus to this "cardiac" region commonly divides into an anterior (B^7a) and a medial ramus (B^7b). The line between the two usually falls in the sulcus of the inferior vena cava.

In 14 per cent of specimens the medial basal bronchus is absent as such, the zone being supplied by one or two accessory rami from the adjacent bronchi—B^8, B*, or B^{10}.

Contrary to Brock, the zone prevailingly occupies two-thirds or more of the anterior margin of the lobe; and contrary to Jackson and Huber it usually extends onto the paravertebral surface.

Because of the high accessible origin of its bronchus, the frequent presence of incisures and the superficial position of this segment, it is deemed suitable for resection.

The *anterior basal segmental bronchus* (B^8) is remarkably constant. It arises from the anterolateral aspect of the basal trunk, a little below B^7. It soon divides into a lateral (B^8a) and a basal ramus (B^8b). The former supplies the area on either side of the interlobar margin below the level of the interfissural crest. The latter supplies the lower part of this sector including the corresponding diaphragmatic area. Usually (50 per cent), B^8b occupies only the lateral one-third of the lower interlobar surface as measured along the inferior margin of the lobe, but in 16 per cent of specimens it extends as far medially as the sulcus of the inferior vena cava. In another 8 per cent it takes over the territory of B^9b. Occasionally (4 per cent), the lateral ramus (B^8a) invades the superior segment.

Because of the high origin of B^8, its fairly constant distribution, and the fact that its medial border is frequently marked by an incisure (36 per cent) it, too, is deemed suitable for resection.

The *lateral basal segmental bronchus* (B^9) arises deep within the lobe and is not considered to be readily resectable. It is absent in 8 per cent of specimens. Typically it divides almost immediately after its origin into a lateral (B^9a) and a basal bronchus (B^9b). The former ramus, when present (86 per cent), sup-

plies the posterior lateral zone adjacent to the subsuperior. When absent, its territory is supplied by B* or B^{8}a. The basal ramus (B^{9}b) supplies the lower posterior lateral sector and the corresponding diaphragmatic area. When absent (8 per cent), its territory is taken over by B^{8}b.

The *posterior basal segmental bronchus* (B^{10}) appears to be a continuation of the lower lobe bronchus—particularly the ramus B^{10}b which extends into the inferior, posteromedial corner of the lobe. Typically, B^{10} breaks up into three major branches. The highest is that which has been already described as the accessory subsuperior, BX*(10). It occurs in 86 per cent of specimens and is reflected upward or backward to supply the subsuperior segment. The two remaining major branches (the mediobasal, B^{10}b, and the laterobasal ramus B^{10}a) pass respectively to the paravertebral and posterior surfaces, and to the corresponding diaphragmatic sectors.

Because of the inaccessibility of its origin, this bronchus, like B^{9}, is not considered to be readily resectable.

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AN ANALYSIS OF VARIATIONS OF THE SEGMENTAL BRONCHI OF THE LEFT LOWER LOBE OF FIFTY DISSECTED, AND TEN INJECTED, LUNGS

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INTRODUCTION

A SURVEY of the literature of the bronchopulmonary segments leaves one with the impression that the left lower lobe of the lung is a somewhat condensed or even defective version of the right lower lobe: that if one understands the pattern of the right side it is necessary to remember only one or two modifications—such as the union of medial and anterior basal bronchi—to comprehend the left.

Such at first was our experience. Soon, however, it began to appear that the left lobe, although capable of being compared with the right, is distinctive throughout. Thus, for instance, the rami of the left superior segment have a different system of branching, the left subsuperior zone is prevailingly posterolateral and not posterior, the left anterior basal and not the left medial basal is the "defective" bronchus, the lateral branch of the left lateral basal contributes to the subsuperior zone, and the left posterior basal, in a third of the specimens, is characterized by a prominent paravertebral ramus.

Hitherto, only Brock (1942 to 1944) seems to have made any detailed study of the variations of the lower lobe segments and he has largely confined himself to describing the varying extent of the segmental bronchi. Our purpose has been to establish the prevailing pattern in a large number of specimens and from this firm and hitherto unestablished base to analyze the significant and puzzling variations which are so frequently encountered.

In general, the terminology of Jackson and Huber (1943) has been employed, supplemented by the system of enumeration of bronchi developed by Boyden (1945). Also, throughout the article, the segmental pattern of the left lower lobe has been compared with that of the right, as recently presented by Smith and Boyden (1949).

MATERIAL AND METHODS

This study is based upon two sets of data: (1) a detailed dissection of fifty preserved specimens (R.M.B.) and (2) colored gelatin injections of ten fresh specimens (F.R.S.).

In the first group, each lobe was dissected from the anterior surface and then accurately drawn. Supplementary sketches recorded the projection of basal

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bronchi on the diaphragmatic surface, the bronchoscopic appearance of the orifices (here inverted), and the sectors supplied by various bronchi—particularly the subsuperiors (for example, text Figs. 5 and 6).

In the second group, fresh specimens were injected with colored gelatins (after the method employed in the study of the right lobe, Smith and Boyden, 1949), and then allowed to harden only one day or so before being dissected and drawn for publication (E.A.B.), for example, Plates 1 to 4.

By means of these two methods—dissection, and injection followed by dissection—it has been possible to establish the prevailing patterns and to display the common variations in the branching and surface distribution of the segmental bronchi.

THE GROSS CONFIGURATION OF THE LEFT LOWER LOBE

The left lower lobe has the shape of a cone, the upper part of which has been compressed. It is divided for descriptive purposes into four surfaces—namely, anterior, costal, paravertebral, and diaphragmatic.

The first two are separated by the sharp interlobar margin (Fig, 4, *B*, and *X*, Fig. 3), the second and third by an ill-defined obtuse margin—the costoparavertebral—which occupies the interval between the heads and tubercles of the ribs (Fig. 4, *B* and *Y*, Fig. 3), and the anterior and paravertebral by the pulmonary ligament and hilum.

The *anterior surface* (middle column of figures, Plate 1) is not a flat plane but exhibits a shallow S-shaped curve—that is, it is molded in such a way that the upper portion faces somewhat laterally and the lower portion somewhat medially. Usually, the lower portion is subdivided into cardiac and interlobar areas by the frenum* of the upper lobe (see Plate 1, No. 52). Usually, as in this plate, the frenum leaves the margin of the hilum between the superior and inferior pulmonary veins, but it may originate anywhere along the pulmonary ligament.† In several of the injected specimens it was coextensive with the pulmonary ligament, and the upper lobe was fused to the cardiac area of the lower lobe. In such cases (Plate 4) this lingular portion of the upper lobe was aerated by small branches of the medial basal bronchus.

The *costal surface* presents a smoothly rounded contour which may be divided arbitrarily—for the purposes of describing segments—into posterior, posterolateral, and lateral sectors (Fig. 4, *B*).

The *paravertebral surface* (Fig. 4, *B*) is molded to the posterior mediastinum and is markedly grooved by the descending aorta. As noted in the foregoing, the costoparavertebral margin is obtuse. For the purpose of describing segments, its position in the plates is indicated by a solid line (Plate 1, left row of figures).

In the course of this study various degrees of fusion between upper and lower lobes were observed. In the literature there is frequent mention of this condition—for example, Kent and Blades (1942) stated that the interlobar

*A term introduced by Churchill and Belsey (1939).

†For more extended discussion, see article by Boyden, E. A.: Cleft Left Upper Lobes and the Split Anterior Bronchus, Surgery, 1949. (In press.)

EXPLANATION OF PLATES

(Sketches rendered for publication by Lawrence B. Benson)

Each plate contains five drawings each, of two injected specimens of the left lower lobe. The large figures at the left depict the costal surface of the lower lobe, the large central ones the anterior surface, and the large figures on the right the dissected anterior surface (each X ¼). In addition, there is a reduced drawing of the diaphragmatic surface (X ⅛) and a small sketch of the orifice of the basal trunk.

Yellow color indicates distribution of superior segmental bronchus (B^6); *white*, the subsuperior zone (B^* and BX^*); *blue* (primarily on anterior surface), the distribution of the medial basal segmental bronchus (B^7); *red*, the distribution of the anterior basal segmental bronchus; *blue* (primarily on costal surface), the distribution of the lateral basal segmental bronchus (B^9); *green*, the distribution of the posterior basal segmental bronchus (B^{10}). *Stippled* areas indicate cut surface of left upper lobe where it is fused with left lower lobe.

Plate 1.—*Specimen 52*, This is characteristically capped by an obliquely placed superior segment (B^6). Typical, also, is the subsuperior zone (B^*), in that it occupies the posterolateral sector of the costal surface. Its extension into the posterior sector is not typical (see Table IV). The basal segments demonstrate the prevailing pattern except that the anterior basal segmental bronchus (B^8) fails to reach the diaphragmatic surface. The two dorsal rami of the posterior basal, labeled (BX^*?), represent accessory subsuperiors which would have been branches of the main trunk of B^{10} had the latter not bifurcated so high (see BX^*[10] in No. 51).

Specimen 51, This is atypical in that B^6a2, the deep branch of the medial ramus of the superior bronchus, has displaced the subsuperior bronchus (B^*) lateralward, on the costal surface. Also the stem of B^{7+8} seems to have rotated medially, leaving that portion of the anterior surface which is usually supplied by B^8a to be taken over by accessory rami (BX^8a) of the subsuperior (B^*) and of the lateral basal (B^9). Accordingly, B^8 (and, to a lesser extent, B^7) supplies a much smaller than usual area.

Plate 2.—*Specimen 56*, This illustrates a subsuperior zone which is supplied by accessory subsuperior bronchi $BX^*(10)$ and $BX^*(9)$, from both the posterior and the lateral basal bronchi respectively. Since these usually arise at the top of their respective bronchi, the tip of the cannula usually projects beyond their orifices leaving the zone uninjected, as in this specimen. This lobe is of further interest in that the superior segment is partially separated from the basal segments by a fissure.

Specimen 58, This very much resembles No. 51 (Plate 1), in that B^6a2 has displaced the subsuperior zone laterally. (In this specimen the zone is supplied by an accessory subsuperior, BX^*, from the posterior basal, B^{10}.) Incidentally, there are two accessory subsuperiors from B^{10}, the second not quite reaching the subsuperior zone. Frequently, as here, a $BX^*(10)$ may arise conjointly with a paravertebral ramus of B^{10}. Also the anterior basal (B^8) encroaches on the medial basal (B^7), but in this case B^8a is present.

Plate 3.—*Specimen 55*, This has a subsuperior zone which is almost a duplicate of that in No. 53 except that it is supplied by two accessory subsuperiors from different sources—namely, high branches of both B^{10} and B^9. The anterior basal (B^8) is represented by both of its rami, but again fails to reach the diaphragmatic surface. Here the medial basal is separated from the other basal segments by a deep fissure, best shown on the diaphragmatic surface. Such fissures are not uncommon (25 per cent).

Specimen 53, Here the subsuperior zone is supplied by a high accessory branch (BX^*) of B^{10} which is almost high enough to be a subsuperior proper. Also, it distributes to the posterior as well as the posterolateral sector of the zone, since B^6a2 is absent. Also in this specimen, the basal ramus of the anterior basal (BX^8b) arises as an accessory branch of the lateral basal (B^9). Therefore B^8 appears to be restricted to a small area on the mid-anterior surface (B^8a).

Plate 4.—These specimens exhibit a fusion of the upper lobe with the lower lobe, the area of fusion extending a varying distance laterally from the whole length of the pulmonary ligament. In each case the thin portion of the lower lingular segment (B^5) was aerated by small accessory branches (BX^5) of the medial basal (B^7). The specimens are also similar in that the basal ramus of B^8 arises as an accessory branch (BX^8b) of the medial basal (B^7).

Specimen 60, Here the subsuperior zone is very extensive, being supplied by a large subsuperior proper (B^*) and two accessory subsuperiors $BX^*(9)$ and $BX^*(10)$. The last of these arises in conjunction with a paravertebral branch of the posterior basal. Also the lateral ramus of the superior segment (B^6c) has what appears to be displaced branches of the superior ramus (B^6b?). This is the "cluster type" of branching noted in Table I.

Specimen 54, This is characterized by three accessory subsuperiors (BX^*), one from the top of the lateral basal (B^9), and two from different levels on the posterior basal (B^{10}). Although one of the last two arises some distance below the other, they both distribute to the same level. The zone as a whole is somewhat depressed by a large B^6a2. In this specimen, the medial ramus of the medial basal arises as an accessory branch of B^9 (see BX^7b).

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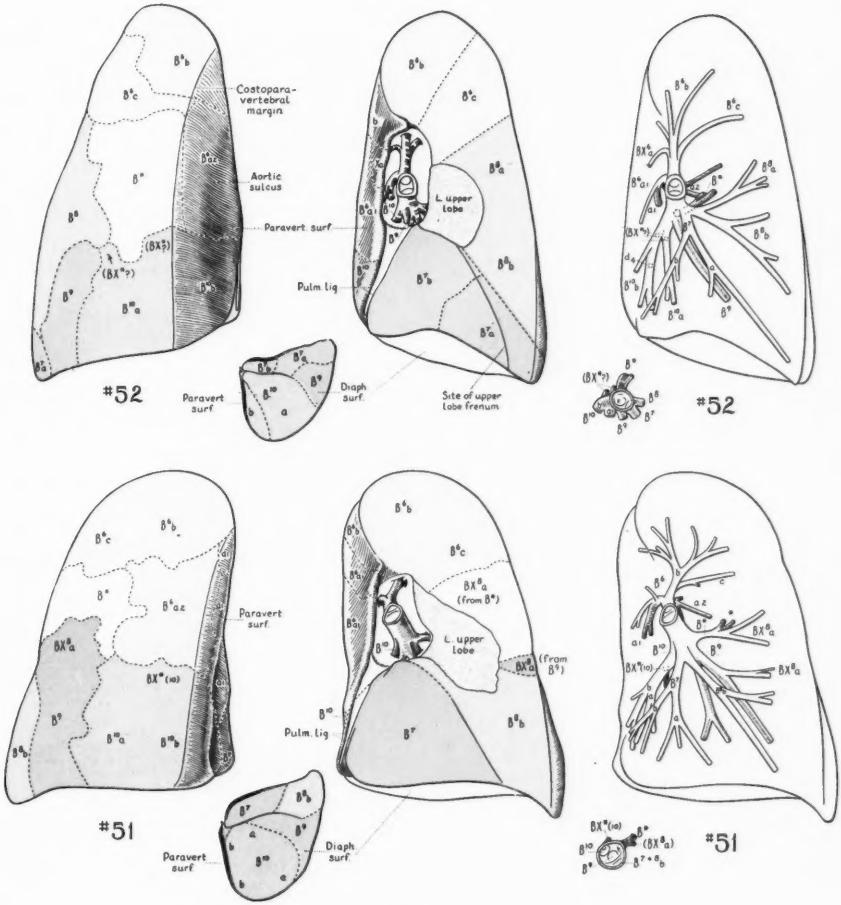

Plate 1.

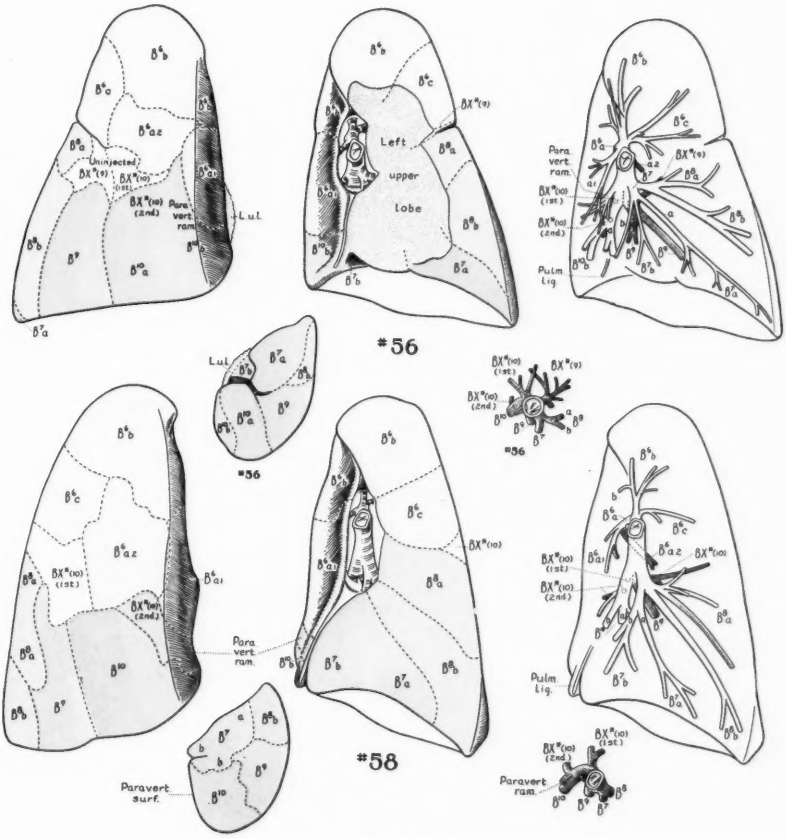
Left upper lobe
Uninjected
Para vert ram.
Paravert surf.
Pulm. Lig.
#56
#58

Plate 2.

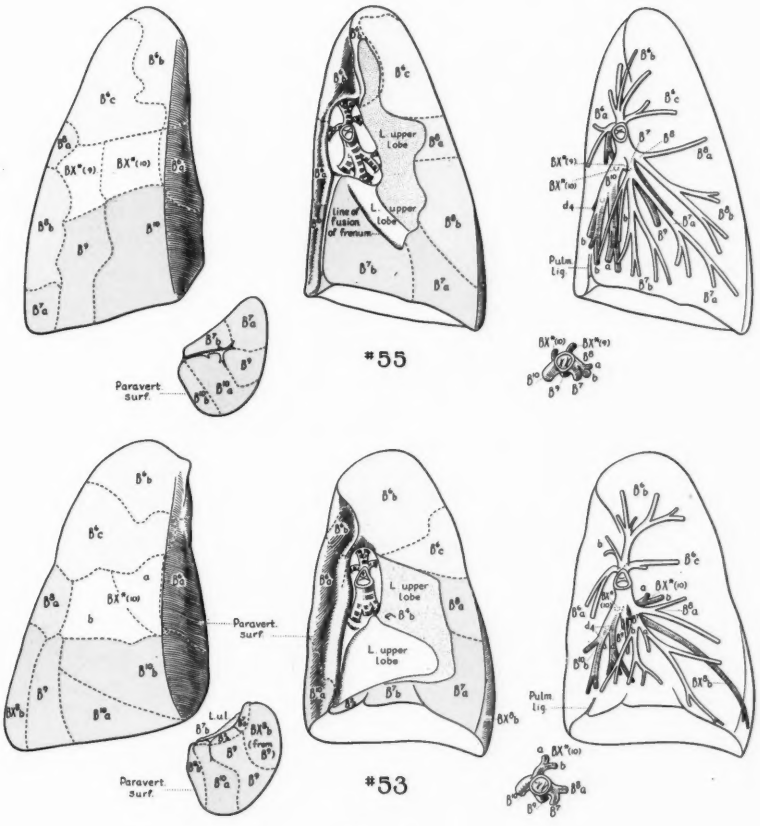
L. upper Lobe
Line of fusion of fremum
L. upper lobe
Pulm. Lig.
Paravert. Surf.
#55
L upper Lobe
L. upper lobe
Paravert. Surf.
Pulm lig.
Paravert. Surf.
#53

Plate 3.

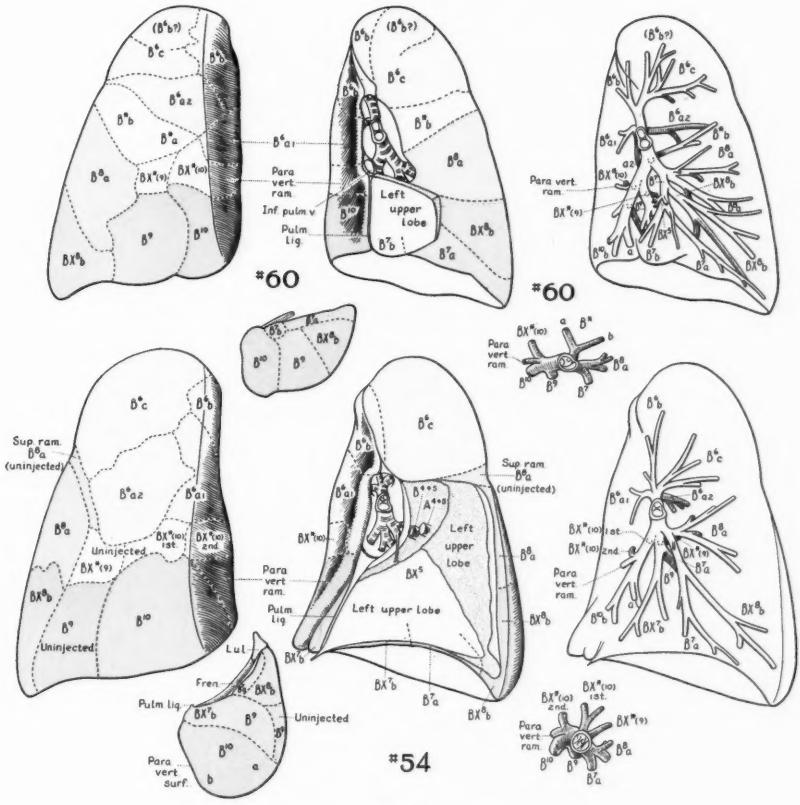
#60
#60
#54
Para vert. ram.
Inf. pulm v.
Pulm lig.
Left upper lobe
Sup. ram. (uninjected)
Uninjected
Para vert. surf.
Fren.
Lul

Plate 4.

fissure was incomplete in about 30 per cent of 277 lungs, being equally common on each side.

THE SUPERIOR SEGMENTAL BRONCHUS (B^6)

The superior bronchus arises just below the upper lobe bronchus in such a position as to complicate resection of the lower lobe, that is, its keel lies only 1 cm. below the keel of the lower lobe bronchus. It is the highest dorsal branch of the lower lobe and therefore has been considered by Brock to be the principal site of lung abscess in this lobe. As on the right side, it breaks up into three constant rami—the medial branch (B^6a) running medially and downward, the superior branch (B^6b) running upward, and the lateral branch (B^6c) coursing laterally and somewhat downward.

In mode of branching the prevailing pattern of B^6 agrees with that of the right side in being bifurcate, but differs in the formula. On the left it usually divides into B^6a and $B^6b + c$ (43 per cent of specimens; Fig. 1, No. 37); on the right it divides into B^6c and $B^6a + b$ (86 per cent).* The percentages indicate that the branching on the left side is more variable than on the right, while the lower and independent origin of B^6a suggests that the paravertebral extent of the bronchus will be much greater on the left side (vide infra).

The frequency of the various types of branching is indicated in Table I. The type recorded in Item I: 2 (see Fig. 1, No. 4) is the one considered most "normal" by Brock (1943). That in Item I: 3 (see Fig. 1, No. 48) is the one described for the left side by Ewart (1889). This is the prevailing pattern on the right side. No one seems to have observed what is termed the "cluster type" (Item I: 4, see Plate 4, No. 60). Here, B^6b is represented by more than one main branch.

Trifurcation of B^6 is relatively uncommon (15 per cent; Fig. 1, No. 55), but not so rare as on the right side (6 per cent).

TABLE I. THE SUPERIOR SEGMENTAL BRONCHUS (B^6)

I. Bifurcates Into:		85%
1. B^6a and $B^6b + c$	(Fig. 1, No. 37)	43%
2. B^6b and $B^6a + c$	(Fig. 1, No. 4)	27%
3. B^6c and $B^6a + b$	(Fig. 1, No. 48)	10%
4. Cluster type		5%
(1) $B^6a + b$ and $B^6b + c$		
(2) B^6a and $B^6b + (B^6b + c)$	(Plate 4, No. 60)	
(3) B^6b and $(B^6b + c) + B^6a$		
II. Trifurcates Into B^6a, B^6b, B^6c (Fig. 1 and Plate 3, No. 55)		15%

The distribution of the superior bronchus is much like that on the right side but differs in at least two particulars. First, in the majority of left lower lobes the superior segment caps the basal segments very obliquely, thus presenting an anatomic hazard to the resection of this segment. (On the right the segment is horizontally placed in 62 per cent.) This is due to the fact that the medial ramus (B^6a) extends downward along the paravertebral surface to

*The statistics for the right side which will be referred to frequently are those published by Smith and Boyden (1949).

within a short distance of the diaphragmatic surface—specifically, it reaches the lower third or less of the paravertebral surface in 57 per cent of specimens (for example, Plate 3, No. 53).*

Second, in 45 per cent of left lower lobes, there is a large posterior branch (B^6a2) which takes over the posterior sector of the subsuperior zone. It is represented occasionally on the right side by a deep branch of B^6a, but on the

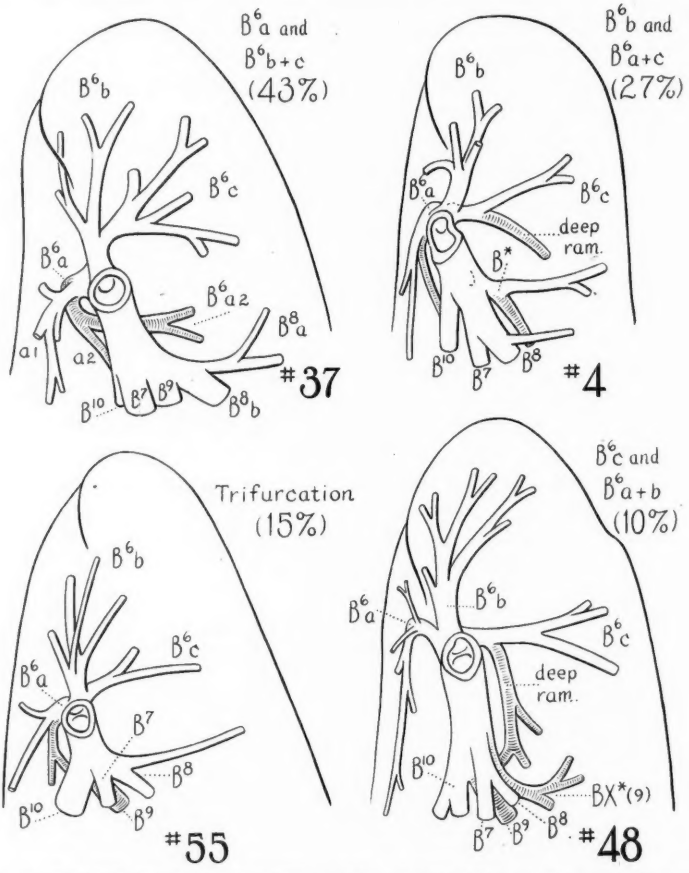

Fig. 1.—Selected drawings illustrating mode of branching of the superior segmental bronchus (X ½). (B^6a, medial ramus; B^6b, superior ramus; B^6c, lateral ramus.) In addition, specimen No. 37 shows a deep branch of ramus *a* (B^6a2) which occurs in 45 per cent of specimens; No. 48 illustrates a deep branch of ramus *c* (*deep ram.*), which occurs in 18 per cent of specimens.

left it is so striking as to merit a special designation (B^6a2, Fig. 1, No. 37 and Plate 4). In certain specimens in which B^6a2 is absent, its territory is taken over by a large, posterior descending branch of B^6c as in Fig. 1, No. 48. (See discussion of subsuperior zone.)

*This finding is, therefore, at variance with the statement of Huizinga and Behr (1940) that "the lower limit of this dorsal segment is always horizontal."

B^6a is of further interest in that in 13 per cent of specimens, part of its paravertebral territory is taken over by an accessory branch of the superior ramus (for example, BX^6a, Fig. 6, No. 23; Plate I, No. 52); in another 2 per cent the lower two-thirds of the paravertebral surface is taken over by a high accessory branch of B^{10}. The distribution of these accessory branches is indicated in the footnote to Table II.

TABLE II. LOWEST EXTENT OF MEDIAL RAMUS (B^6a) ON THE PARAVERTEBRAL SURFACE

Two-thirds or more of distance to diaphragm (Plate 3, No. 53)	57%*
Halfway to diaphragm (Plate 4, No. 60)	37%
One-third of distance to diaphragm	3%
Fails to reach paravertebral surface and is replaced by a branch of B^6b	3%

*In 8 per cent the upper portion of paravertebral surface is replaced by an accessory branch of B^6b. Since this occurs altogether in only 15 per cent of specimens it would seem that when the medial ramus descends the lowest there is a tendency for its upper territory to be taken over by B^6b.

Discussion of this segment should not be concluded without mentioning the occurrence of a notch on the lateral side of the interlobar margin of the lobe which marks the separation of the superior from the basal segments in 5 per cent of specimens (Fig. 6, No. 8; Plate 2, No. 56) and of an extensive fissure recently observed in an autopsy specimen. This is a much lower frequency than was reported by Dévé in his study of 180 infants (14 per cent on the left, 30 per cent on the right, 12 per cent bilateral). Pierret, Coulouma, Breton, and Devos (1938) did not give separate figures for the two sides but reported the occurrence of the fissure in 20 per cent of specimens. Nelson (1934) considered that this inconstant fissure provided supporting evidence for his classification of the superior segment as a separate lobe, but it would seem that the fissure is no longer necessary to the argument.

THE SUBSUPERIOR BRONCHUS (B^*)

On the right side the subsuperior bronchus has been identified as the second dorsal ramus of Aeby, that one which arises from the stem bronchus between the first dorsal or superior bronchus (B^6) and the origin of B^9 (B^*, Fig. 2, *A*). On that side, it usually supplies the region which occupies the interval between the superior and the posterior basal segments in the posterior sector of the costal surface. On the left side, we will show that it arises from the same portion of the stem bronchus (B^*, Fig. 2, *B*), is distributed to the same horizontal level, but that it supplies the posterolateral sector.

Neil, Gilmour and Gwynne (1937) were the first modern investigators to call attention to this "site of localized pathological change—an anatomical gap in the distribution between the apical [B^6] and paravertebral [B^{10}] bronchi." When this bronchus was absent they noted that the "subapical segment" was supplied by a branch of the posterior basal (B^{10}). Smith and Boyden (1949) confirmed these observations and named the two types of bronchi that supply this intermediate zone the *subsuperior proper*, B^*, and the *accessory subsuperior*, $BX^*(10)$.† Because of the prevailingly dual and variable bronchial supply,

†Interpolation of the letter "X" indicates that the bronchus supplying a given zone arises from another portion of the bronchial tree than is customary. The figure in parentheses tells its new site of origin. Thus "$BX^*(10)$" indicates that the subsuperior zone is supplied by a high branch of B^{10} instead of by the second dorsal branch of the stem bronchus.

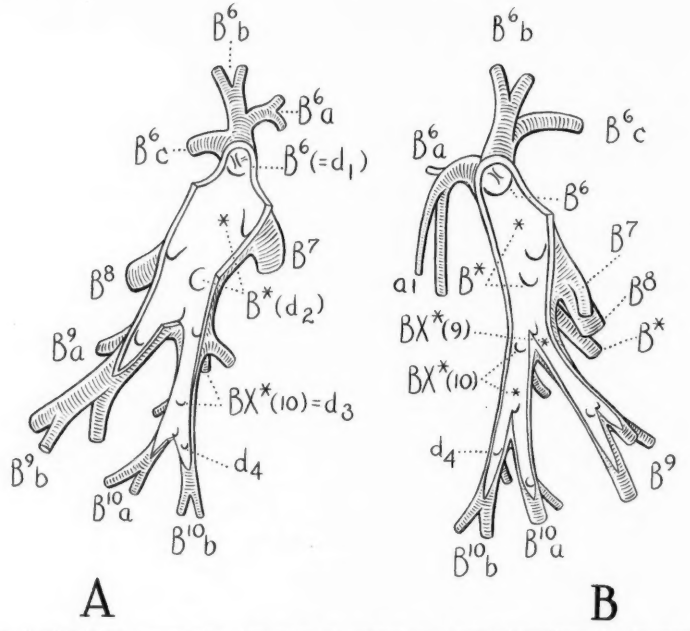

Fig. 2.—Sketches comparing the sites of origin of dorsal branches of the stem bronchus in left and right lower lobes. *B*, drawing of left side (specimen No. 50). *A*, drawing of right side after Smith and Boyden (1949). The specimen in *A* has five dorsal rami; the site of a possible sixth is noted by an asterisk. These have been grouped into four levels, corresponding to the four dorsal bronchi postulated by Aeby, in 1880. The first level (d_1) is the site of the superior segmental bronchus (B^6); the second (d_2), the site of the subsuperior proper (B*); the third (d_3), the site of the accessory subsuperior (BX*). The orifices of corresponding dorsal bronchi on the left side are shown in B. The principal difference between the two sides as that on the left the dorsolateral branch of B^9 (BX*[9]) also participates in the subsuperior zone (see discussion in text).

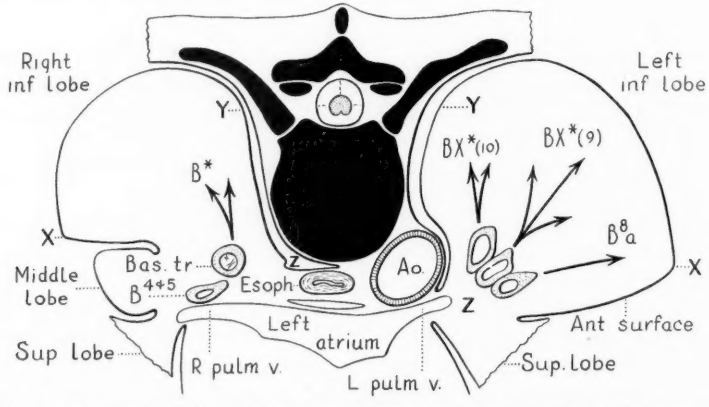

Fig. 3.—Transverse section of the lower lobes of an intact thorax to illustrate the differing direction of the subsuperior bronchi on the two sides. On the right, the subsuperior proper (B*) distributes primarily to the posterior sector of the costal surface; on the left, it may distribute to the posterior sector (see BX* from B^{10}), but prevailingly it has a posterolateral distribution (see BX* from B^9). BX*(10) and BX*(9) are left accessory subsuperiors, arising just beneath the left subsuperior proper (B*), which does not appear in this figure. *X*, interlobar margin of lower lobe; *Y*, costoparavertebral margin; *Z*, hilum (or, lower down, the pulmonary ligament).

they did not follow Neil in giving this zone the status of a segment, or numbering it—hence the noncommittal designation B*.

On the left side, this intermediate zone is equally conspicuous and clinically significant, but the bronchi supplying it are more numerous. In a later article (1939) Neil and his co-workers stated that the subapical bronchus [B*] "may be absent on the left side, in which case we have found a branch going to its customary distribution arising from the axillary basal [B^9] or from the posterior basal [B^{10}]." With this conclusion we would also agree, but it would seem that the evidence supporting it has yet to be presented.

In the first place, analysis of the 60 specimens indicates that on the left side, as contrasted with the right, the subsuperior proper (B*) always distributes to at least the posterolateral sector and most commonly to both the posterolateral and lateral sectors of this intermediate zone (see Table IV, Item IV), whereas on the right side it is distributed primarily to the posterior sector (B*, Fig. 3). Furthermore, the angle it makes with the anterior surface is prevailingly 40 to 45 degrees (23 per cent; see Table IV, Item III: 1).

For this reason, both the uppermost dorsal branches of B^9 and B^{10} that enter into the composition of the subsuperior segment must be considered acces-

TABLE III. OCCURRENCE OF SUBSUPERIOR BRONCHI (B* AND BX*)

I. Frequency of Occurrence in 60 Left Lower Lobes†				*In 50 rt. lower lobes‡*
1. Origin exclusively from level d_2 (as branch of main lower lobe bronchus between the superior and basal bronchi)			7%	14%
2. Origin exclusively from level d_3 (as an accessory subsuperior, BX*(10) or BX*(9)			71%	38%, BX*(10)
3. Origin from both levels in same specimen			22%	48%
			100%	100%
II. Detailed Analysis				
1. Subsuperior represented by single bronchus			22%	50%
(1) Arising at d_2 level (as B*)		7%		(14%)
(2) Arising at d_3 level (as BX*)		15%		(34%)
(a) From top of B^{10}	8%			
(b) From top of B^9	7%			
2. Subsuperior represented by two bronchi			61%	34%
(1) One from level d_2(B*) and one from level d_3(BX*)		13%		(32%)
(a) B* and BX*(10)	11%			
(b) B* and BX*(9)	2%			
(2) Two from level d_3 (BX*)		48%		(2%)
(a) BX*(9) and BX*(10)	43%			
(b) Two BX*(10)	5%			
3. Subsuperior represented by three bronchi			17%	14%
(1) Two from level d_2(B*) and one from level d_3, BX*(10)		2%		
(2) One from level d_2(B*) and two from level d_3, BX*(10) and BX*(9)		7%		
(3) Three from level d_3 Two BX*(10) and one BX*(9)		8%		
4. Subsuperior represented by four bronchi (two from each level)			0%	2%
			100%	100%

†Percentages in round numbers.
‡Data from Smith and Boyden (1949).

sory subsuperior bronchi. These are accordingly designated BX*(10) and BX*(9). (See Fig. 3.)

The origins of the various subsuperior bronchi are indicated in Fig. 2, *B*. This is a sketch of an actual specimen (No. 50) showing two subsuperiors present—namely, one arising from level 2 of Aeby (B*), and a second, BX*(10), from the top of the posterior basal (B^{10}), that is, from level 3 of Aeby. Three other sites where these may occur are indicated on the bronchial tree by asterisks —namely, a subsuperior proper (in level 2) which arises above the orifice of B^7, an accessory subsuperior (in level 3) which arises at the top of the lateral basal bronchus, BX*(9), and an accessory subsuperior (from the lowest part of level 3) the second BX*(10). (The latter site appears to develop an accessory subsuperior only when there are two BX*[10] in the same specimen.) In Plate 4 (No. 60) a subsuperior zone is shown which is supplied by three bronchi—a subsuperior proper, B*, and two accessory subsuperiors, BX*(10) and BX*(9).

A summary of the number of bronchi supplying this zone may be found in Table III, together with corresponding figures from the right lower lobe.

A comparison of the percentages for left and right lower lobes shows striking differences. First, the subsuperior proper (B*) occurs much less frequently on the left side (29 per cent) than on the right (62 per cent: Item I: 1 + 3). Second, the number of specimens in which there are two subsuperiors (Item II: 2) is much greater on the left (61 per cent) than on the right (34 per cent), and this is due primarily to the participation on the left of BX*(9), the high lateral branch of B^9.

At this point, the question may be raised as to why the subsuperior zone on the left side is primarily posterolateral (and lateral) and not posterior. One answer would seem to be that the posterior sector is pre-empted by the deep posterior branch of the superior segment (B^6a2) in 45 per cent of specimens (see Plates 4, and 2), and by the deep ramus of B^6c (Fig. 1, No. 48) in another 18 per cent. Thus, if one did not realize the differing orientation of the subsuperior zone on the left, one might assume that a lung abscess in B^6a^2 involved the subsuperior bronchus.

1. *The Subsuperior Proper* (*B**).—The subsuperior bronchus proper arises from the basal stem at either of two levels, both within the limits of what we

TABLE IV. POSITION AND ORIENTATION OF SUBSUPERIOR PROPER (B*)

I. Level on stem bronchus	
1. Above the origin of B^{7+8} (Fig. 5, *G*)	5%
2. From the common stem of B^{9+10} (Plate 1, No. 52)	24%
II. Position of orifice on circumference	
1. Posterior (or slightly posterolateral) (Fig. 5, *H*)	12%
2. Posterolateral (Plate 1, No. 51)	12%
3. Lateral (or slightly posterolateral) (Fig. 7, *D*)	5%
III. Angle made with anterior surface	
1. 40-45 degrees (i.e. posterolateral) (Plate 1, No. 51)	23%
2. 80-100 degrees (i.e. posterior) (Fig. 5, *H*)	3%
3. 20-30 degrees (i.e. almost lateral) (Fig. 7, *D*)	3%
IV. Distribution on costal surface	
1. Posterolateral and lateral (Plate 1, No. 51)	14%
2. Posterior, posterolateral, and lateral (Plate 4, No. 60)	10%
3. Posterolateral only	3%
4. Posterior and posterolateral (Plate 1, No. 52)	2%

have defined as the d_2 level. In 5 per cent it originates from the highest position indicated in Fig. 2, *B*, that is, above the origin of $B^{7\text{ and }8}$; in the remaining 24 per cent it arises from the common stem of B^{9+10}.

Its position on the circumference of the basal stem is varied. In 12 per cent its orifice is posterior (or slightly posterolateral); in another 12 per cent it is posterolateral; and in 5 per cent it is lateral (or slightly posterolateral). (See Table IV, Item II.) Neil and co-workers (1939) "found it situated dorsally, medially, laterally and twice ventrally." Neil's medial subsuperior is presumably one of the high accessory subsuperiors, BX*(10), which occasionally arises medially in conjunction with a high paravertebral branch of B^{10} (Fig. 6, *E* and Plate 4, No. 60). Neil's ventral subsuperior could have been a high B^8a with posterolateral branches.

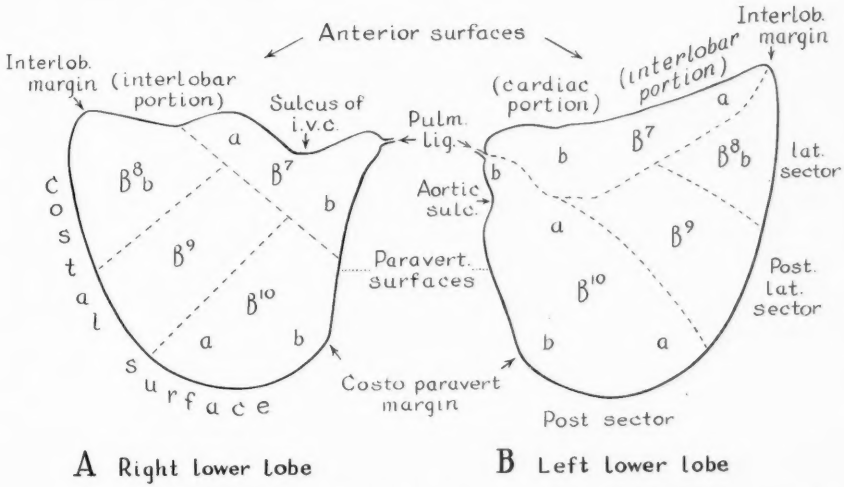

Fig. 4.—Diagrams illustrating typical arrangement of basal segments in a cross section of the lower lobes made just above the diaphragm, and viewed from below. *A, right lower lobe* (after Smith and Boyden). Note that the medial basal segment (B^7) is placed athwart the other segments and that the latter overlap each other as postulated by Melnikoff. *B, left lower lobe,* illustrating shift in position of B^7; on this side it lies wholly lateral to the pulmonary ligament, failing to reach the paravertebral surface. That site is now occupied by the posterior basal segment (B^{10}); nevertheless, B^7 is as extensive as on the right. Note that the basal ramus of the anterobasal segment (B^8b) supplies the lateral sector of the costal surface; that the lateral basal segment (B^9) supplies the posterolateral sector; and that the posterior basal (B^{10}) supplies the posterior sector and the paravertebral surface.

This brings out the point that it is often hard to distinguish between a laterally originating B* (for example, Fig. 7, No. 4) and the lateral ramus of the anterior basal bronchus, B^8a (for example, Fig. 7, No. 41). In the former case, a lateral B* distributes primarily to the posterolateral sector of the subsuperior zone but it has branches that take over the upper anterolateral zone (BX^8a). Superficially the two specimens look somewhat alike, but when the orifice of B* is noted (Fig. 7, *D*), the bronchus is seen to arise more posterolaterally, behind B^9.

In Fig. 5, specimen No. 39 is somewhat similar, but here (Fig. 5, *H*) the orifice of the subsuperior bronchus is posterior, that is, behind all basal bronchi.

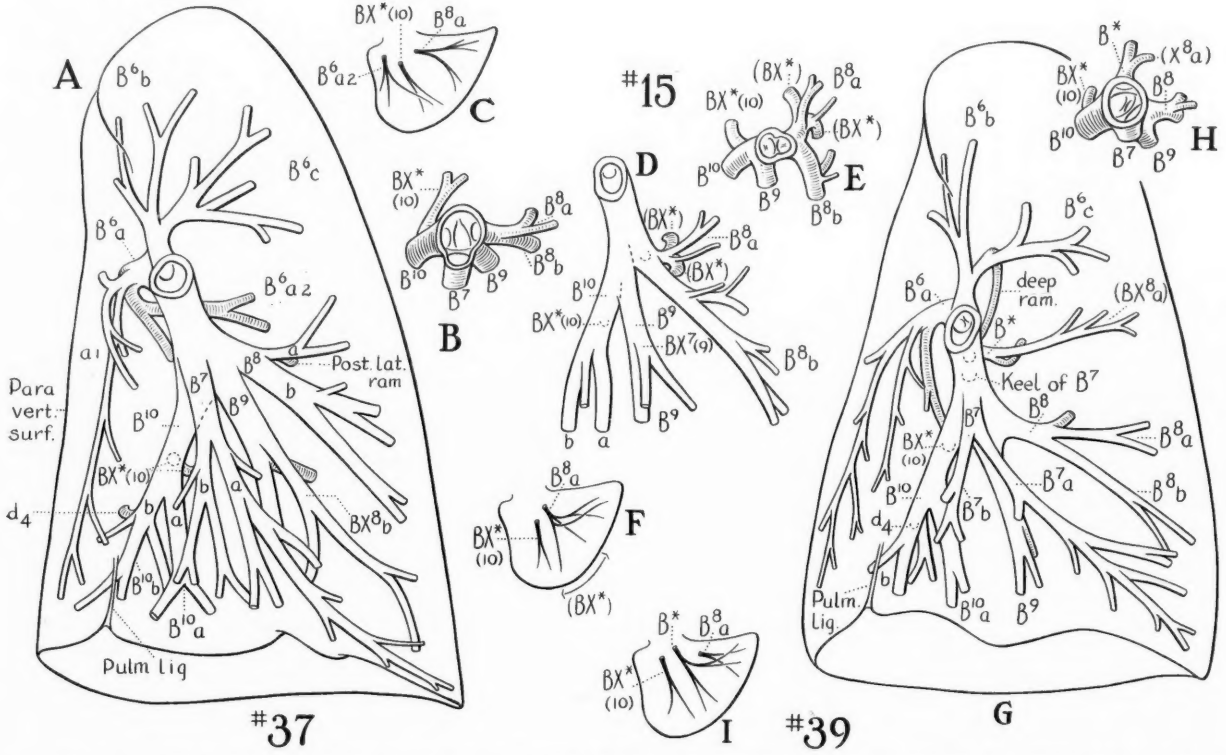

Fig. 5.—Drawings of the bronchial trees of three preserved left lower lobes (X½) to show (1) transitional patterns between B^8a and B^*; (2) anomalous origins of B^7 and B^8. *A, B, C* anterior dissection, orifice of basal trunk, and cross section, respectively, of specimen No. 37. B^8 is typical except for a posterolateral ramus that simulates the distribution of a subsuperior; B^7 arises separately, as in the right lower lobe; B^9 takes over part of the territory of B^8b (see BX^8b). *D, E, F* are corresponding views of specimen No. 15. B^8a is atypical in that it has an accessory subsuperior rami (BX^*). B^7 (as BX^7) arises anomalously from the lateral basal (B^9). *G, H, I* are corresponding views of specimen No. 39. B^* (the subsuperior) takes over part of the territory of the lateral ramus of B^8 (see BX^8a). B^7 again arises separately from B^8.

It is a subsuperior both because of its origin and because of its distribution; yet, again there is a secondary distribution to the B^8a zone.

In specimen No. 15 (Fig. 5) the situation is reversed. Here the corresponding bronchus has a lateral origin in common with B^8b and therefore its main stem must be designated B^8a. The deeper portion of it, which distributes to the subsuperior zone, may be called a displaced subsuperior, BX*(8). In specimen No. 37, B^8a makes a lesser contribution to the subsuperior zone (see Fig. 5, *C*) and therefore is merely designated B^8a.

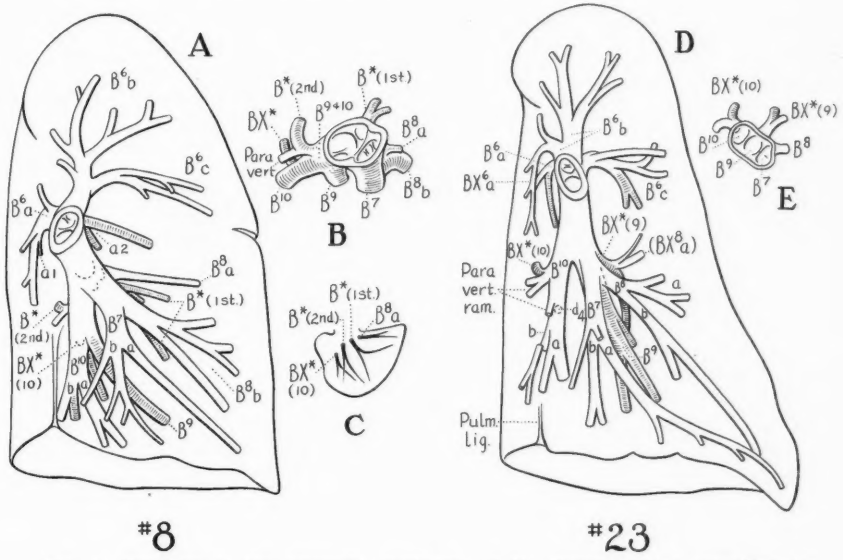

Fig. 6.—Drawings of two preserved specimens (X 2/5) illustrating the prevailing (bifurcate) and atypical (trifurcate) mode of division of the basal trunk. *A*, *B*, *C*, are anterior dissection, orifice of basal trunk, and cross section, respectively, of specimen No. 8. The trunk bifurcates into an anterior (B^{7+8}) and a posterior division (B^{9+10}). *D*, *E*, are anterior dissection and orifice, respectively, of specimen No. 23, showing a trifurcation into three stems, B^{7+8}, B^9, and B^{10}. Incidentally, these figures show how a high division of B^9 and B^{10} may convert a subsuperior proper (B^*, 2nd, of No. 8) into an accessory subsuperior (BX*[10], of No. 23).

Brock also realized the difficulty of describing these transitional forms. His solution for handling a laterally originating B* like that in Fig. 7 (No. 4), is to call it a "supernumerary axillary anterior basic bronchus," as if it were the axillary (lateral) ramus of B^8 which had arisen higher than usual—that is, from the main stem. In his own words he preferred "to restrict the name subapical [subsuperior] to the one inconstant but commonest, posterior bronchus and to name and describe any others separately (see axillary branches, p. 63)." However, he apparently did not realize that the one inconstant subsuperior bronchus arising from the lower lobe stem on the left side distributes to the posterolateral sector. Thus, if his preference were followed, most left subsuperiors would have to be called supernumerary axillary middle basals, which would destroy the useful concept of an intermediate zone interpolated between

the superior and basal segments. Furthermore, Brock's use of the term axillary to cover bronchi distributing to lateral, posterolateral, and posterior sectors of the lower lobe, while having perhaps a certain clinical value, is not precise enough to be of use in analyzing patterns.

2. *The Accessory Subsuperior, BX*(10).*—An accessory subsuperior arises as a single branch of the posterior segmental bronchus (B^{10}) in 71 per cent of specimens and as two separate rami in 13 per cent. When single its keel lies typically within a distance of 12 mm. below the keel separating B^9 and B^{10}. When two are present they originate at the levels shown in Fig. 2, *B*, but may distribute to the same surface level (Plate 4, No. 54). In view of the marked

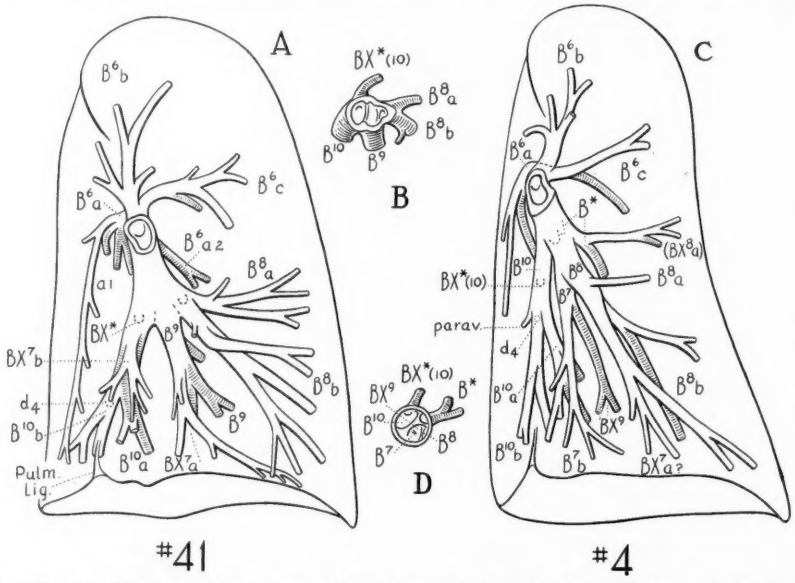

Fig. 7.—Sketches of two preserved specimens (X ½) illustrating bizarre origins of the medial and lateral basal bronchi, B^7 and B^9. *A, B,* are anterior dissection and orifice of basal trunk of No. 41. B^7a (as BX^7a) arises as an accessory branch of B^9. B^7b (as BX^7b) originates as an accessory branch of B^{10}. *C, D,* are corresponding views of specimen No. 4. B^9 (as BX^9) arises as an accessory branch of a deep-lying B^7.

lateral shift of B* on the left side, it is interesting to note that BX*(10) also distributes to a posterolateral sector in one-half of the specimens in which it occurs (Fig. 5, *C*, No. 37). This is in marked contrast to its behavior on the right side, where it invades the posterolateral sector in only about one-sixth of the cases in which it is the only subsuperior present. The percentages for the left side are given in Table V.

3. *The Accessory Subsuperior BX*(9).*—An accessory subsuperior arises in 67 per cent of specimens from near the top of the lateral basal segmental bronchus (B^9). Like BX*(10) its keel lies typically within 12 mm. of the distance below the keel of B^9 and B^{10}. Prevailingly it distributes to the posterolateral sector (see Table VI) and, as would be expected, makes an angle of about

TABLE V. DISTRIBUTION OF BX*(10)

I. Sector	
1. Wholly posterior (Plate 4, No. 60)	41%
2. Posterior and posterolateral (Plate 3, No. 53)	26%
3. Posterolateral only	16%
4. Posterolateral and lateral (Plate 2, No. 58)	1%
II. Angle With Anterior Surface of Lobe	
1. 75-100 degrees (Plate 2, No. 58)	61%
2. 45-50 degrees (Fig. 7, *B*)	23%

45 degrees with the anterior surface in the majority of specimens (see Fig. 3). However, it has a strong tendency to shift laterally. In this respect it resembles the subsuperior proper (see Table IV). It is homologous with B^9a of the right lobe, but on the right side B^9a does not usually participate in the subsuperior zone, since the subsuperior zone of the right lung is primarily restricted to the posterior sector.

TABLE VI. DISTRIBUTION OF BX*(9)

I. Sector	
1. Posterolateral only (Plate 4, No. 54)	40%
2. Posterolateral and lateral (Plate 3, No. 55)	14%
3. Posterior, posterolateral, and lateral	5%
4. Posterior and posterolateral	8%
II. Angle With Anterior Surface of Lobe	
1. 40-60 degrees (Plate 2, No. 56)	45%
2. 20-35 degrees	12%
3. 70-90 degrees (Plate 3, No. 55)	10%

To summarize this sector, it has been pointed out that the criterion for defining the subsuperior zone has been the prevailing distribution of the subsuperior bronchus proper (B*). On the right side, it is prevailingly posterior, and on the left, prevailingly posterolateral. Any additional bronchi that regularly distribute to this zone have therefore been called accessory subsuperiors, notably BX*(10) on both sides and BX*(9) on the left. Certain other extraneous branches that sometimes invade the posterior or lateral sector of this zone—such as the more posterior rami of B^6a, B^6c, and B^8a—might perhaps be included. Indeed, Ewart describes "a central lozenge-shaped bronchial interspace" in the left lower lobe formed by "the lesser posterior horizontal [BX*(10)] . . . together with the external mammary [B^8a] and the midaxillary bronchus [BX*(9)]." However, it seems sufficient to point out that such extraneous branches may invade this zone and to note the criteria by which they may be distinguished from the true subsuperior and the more frequently occurring accessory subsuperiors.

THE BASAL BRONCHI

The subdivision of the basal trunk below the level of the superior bronchus (B^6) is somewhat different on the left side from that on the right. On the left the medial basal segmental bronchus (B^7) arises more anterolaterally and usually in conjunction with the anterior basal (B^8), instead of being the highest and most medial basal bronchus. For this reason various recent authors have considered B^7 and B^8 collectively as a single segmental bronchus. Here, for purposes of description, at least, they will be treated separately.

TABLE VII. MODE OF BRANCHING OF BASAL TRUNK*

I. Basal Trunk Bifurcates			87%
1. Into B^{7+8} and B^{9+10} (Fig. 6, No. 8)		67%	
2. Into bizarre patterns		20%	
(1) B^7 and B^8 + (B^{9+10}) †(Fig. 5, No. 37)	8%		
(2) B^{10} and B^8 + (B^{7+9}) (Fig. 7, No. 4)	7%		
(3) B^7 and (B^{8+9}) + B^{10} (Fig. 5, No. 39) B^8 and (B^{9+7}) + B^{10} (Fig. 5, No. 15) B^{7+8a} and $B^{8}b$ + ^{10} (B^9 absent)	5%		
II. Basal Trunk Trifurcates‡			
Into B^{7+8}, B^9, and B^{10} (Fig. 6, No. 23)			13%

*The subsuperiors are ignored in this classification, together with such minor branches as high paravertebral branches of B^{10}.

†Resembles typical pattern of right basal trunk.

‡Counted as a trifurcation when keels were not more than 4 mm. apart. In another 7 per cent the keels were a few millimeters further apart.

Below the point of origin of B^{7+8}, the basal trunk continues as the common stem of the lateral and posterior segmental bronchi (B^9 and B^{10}, respectively). In other words the prevailing pattern, contrary to what has hitherto been supposed, is a bifurcation of the basal trunk into B^{7+8} and B^{9+10} (Fig. 6, No. 8).* Trifurcations occur in only 13 per cent (Fig. 6, No. 23). The percentages and varying combinations are recorded in Table VII.

THE MEDIAL BASAL SEGMENTAL BRONCHUS (B^7)

The medial basal bronchus arises in common with the anterior basal (B^8), or one of its two branches, in 87 per cent of specimens (Table VIII). This short stem emerges from the anterolateral aspect of the basal trunk. Prevailingly (47 per cent), B^7 lies somewhat anterior to B^8 (Fig. 6); frequently (35

TABLE VIII. THE MEDIAL BASAL BRONCHUS (B^7)

I. Origin			
1. Arises in common with B^8 (or one of its rami)*			87%
(1) B^7 lies in plane anterior to B^8	47%	(Fig. 6)†	
(2) B^7 and B^8 lie in same plane	35%	(Plate 1, No. 52)	
(3) B^7 lies deep to B^8	5%	(Fig. 7, No. 4)	
2. Arises separately from B^8 on anterior side of main stem (Fig. 5, No. 37, No. 39)			10%
3. Absent as such but occurs as accessory ramus of B^9 or as rami of B^9 and B^{10} (Fig. 5, No. 15; Fig. 7, No. 41)			3%
II. Distribution			
1. Supplies the whole width of the inferior portion of anterior surface (Plate 3, No. 53)			55%
2. Supplies medial ½ to ⅔ of inferior portion of anterior surface (Plate 2, No. 58)			20%
3. Supplies the whole inferior portion of anterior surface and also a sector of costal surface			17%
(1) By spreading of its branches (Plate 3, No. 55)		10%	
(2) By virtue of accessory rami (BX^9 or BX^8b) (Plate 4, No. 60)		7%	
4. Supplies medial ½ to ⅔ of inferior portion of anterior surface and also a costal sector (BX^9 or BX^8b) (Fig. 7, No. 4)			5%
5. B^7 absent as such; represented by accessory rami of adjacent bronchi (Fig. 7, No. 41)			3%

*In one specimen B^7 was represented in its usual place by only one of its two rami, B^7a; the other ramus, B^7b was displaced, arising as an accessory branch of B^9 (plate 4, No. 54).

†In one specimen B^7 and B^8a arose together, more anteriorly than B^8b.

*Davis (1929) found a trifurcate pattern in 53 per cent of 32 lungs.

per cent), it lies in the same plane (Plate 1); rarely (5 per cent) it lies deep to B^8 (Fig. 7, No. 4).

Of special interest are the 10 per cent of specimens in which B^7 arises separately from the basal trunk thereby simulating the appearance of its homologue in the right lung. Two such specimens are displayed in Fig. 5 (No. 37 and No. 39). Dévé (1900) was the first to describe this pattern and De Pablo (1940), Hagens (1943), and Foster-Carter (1946) have reported it more recently.

In 3 per cent, B^7 is absent from its usual position, arising as an accessory ramus of B^9 or as rami of B^9 and B^{10} (Fig. 5, No. 15; and Fig. 7, No. 41).

Since the typical pattern in the left lung is characterized by a common origin of B^7 and B^8, many different views have been held with respect to the nature of B^7. Some authors (Aeby, Kramer and Glass, Nelson, Neil, etc.) stated that B^7 is not represented in the left lower lobe. Others (Pierce and Stocking, 1939, Foster-Carter, 1942, Adams and Davenport, 1942, Jackson and Huber, 1943, Brock, 1943, etc.) relegate it to the status of a subsegmental bronchus or less. Only a few investigators, Lucien and Weber (1936), De Pablo (1940), Appleton (1944), etc., maintain that B^7 is a distinct segmental bronchus.

On the basis of its distribution in the sixty specimens presented in this study, we have reluctantly come to the conclusion that it should be considered as a major bronchus. The mere fact that it arises in conjunction with B^8 is no more relevant than the fact that the segmental bronchi of the middle lobe have a common stem.

When the typical distribution of the left medial basal bronchus is compared with the right medial (Fig. 4), it will be noted that the two bronchi aerate an equally large zone. The difference is that on the left B^7 has shifted laterally. Instead of supplying part of the paravertebral surface and part of the anterior surface, it has withdrawn from the paravertebral surface (contrary to Lucien and Weber) and extends from the pulmonary ligament to the interlobar margin. Finally, analysis of the anterior basal bronchus (B^8) shows that it and not B^7 is the defective bronchus (vide infra).

The details of the distribution of B^7 are recorded in Table VIII. In 55 per cent of specimens (Item II: 1) it aerates the whole inferior portion of the anterior surface of the lobe, as measured along the inferior margin (Plate 3, No. 53). In a smaller group (20 per cent: Item II: 2) it supplies only the medial one-half to two-thirds of the lower anterior surface (Plate 2, No. 58). In the remaining 22 per cent in which B^7 is present (Items II: 3 and 4) it invades the costal surface, either by spreading of its branches (Plate 3, No. 55) or by virtue of accessory rami (Plate 4, No. 60).

From another standpoint, it also deserves an independent status—namely, its resectability. Not only is the bronchus available, being visible at the inferior margin of the hilum (Plate 3, No. 55) but in 25 per cent of specimens its medial border (at the pulmonary ligament) is marked by a fissure which extends well into the diaphragmatic surface (Plate 2, No. 56 and Plate 3, No. 55). In addition, there is usually a notch at that point.

Finally, mention should again be made of those specimens in which the left upper lobe is fused to the cardiac surface of the lower lobe. In some instances, B^7 may aerate this adherent laminar portion of the upper lobe (Plate 4, No. 54 and No. 60); in others, the latter is supplied by a lingular bronchus (Plate 2, No. 56).

THE ANTERIOR BASAL SEGMENTAL BRONCHUS (B^8)

The anterior basal segmental bronchus is the least constant of the basal bronchi of the left lower lobe and thus presents a marked contrast to its homologue of the right side; for in 27 per cent of specimens it is defective either in origin—that is, one of its two rami arises as an accessory branch of another bronchus—or it fails to reach the diaphragmatic surface.†

Typically (in 70 per cent of specimens), the anterior basal arises with the medial basal (B^7) through the medium of a short common trunk which may be seen at the lower end of the hilum arising high up on the anterolateral aspect of the basal trunk (Plate 2, No. 58). In another 13 per cent (Table IX, Item I: 2), it arises alone at the site of the common trunk (Fig. 7, No. 41). In the remaining 17 per cent either its basal ramus (B^8b) or its lateral ramus (B^8a) is absent as such, being represented by an accessory branch of another bronchus (Item I: 2, [2] and [3]). In general the accessory lateral rami arise from the subsuperiors, BX*(9) or B* (Plate 1, No. 51), and the accessory basal rami take origin from either the medial or lateral basal bronchi, B^7 and B^9 (Plate 4, No. 54; Plate 3, No. 53). In addition, rami from adjacent bronchi may spread into the territory of B^8, crowding the latter, for example, out of the diaphragmatic surface (Plate 1, No. 52).

TABLE IX. THE ANTERIOR BASAL BRONCHUS (B^8)

I. Origin		
1. Arises from a short common trunk with B^7		70%
2. Atypical origin		30%
(1) B^8 arises alone from basal trunk in its usual place† (Fig. 5)	13%	
(2) B^8a only arises with B^7	8.3%	
BX^8b from basal trunk		
BX^8b from B^9 (Plate 3, No. 53)		
BX^8b from B^7 (Plate 4)		
(3) B^8b only arises with B^7	8.3%	
BX^8a from BX*(9)		
BX^8a from B* (Plate 1, No. 51)		
II. Distribution		
1. Reaches diaphragmatic surface (Plate 2, No. 56)		83%
2. Fails to reach diaphragmatic surface (Plate 3, No. 55)		17%

†In one specimen B^8 arose in conjunction with B^9.

The normal distribution of B^8 on the anterior surface is best illustrated by Plate 2, No. 56. The lateral ramus (B^8a) supplies the upper half (upper third to two-thirds) of that part of the anterior interlobar surface which lies below the superior segment, together with the adjacent lateral costal surface (Plate 2, No. 58). Since the zone between B^6 and B^8b is at the same level as the interpolated subsuperior segment, the subsuperiors (B*, and BX* from B^9)

†In 17 per cent, it is defective in number of branches (Table IX, Item I:2), and in 17 per cent it fails to reach the base of the lung (Item II:2). Since seven of the 34 per cent are atypical in both respects, there remains a total of 27 per cent defective segments.

frequently send accessory branches to the B^8a zone (Fig. 6, No. 23) and sometimes take it over completely (Plate 1, No. 51).

The basal ramus (B^8b) typically supplies the lower lateral half (lower third to two-thirds) of the interlobar surface, that is, lateral to a line drawn from the inferior margin of the hilum to the lateral inferior angle of the lobe (Plate 2, No. 56). In only 27 per cent of specimens does it invade the area medial to this line (Plate 2, No. 58). Its typical diaphragmatic distribution is shown in Plate 2, No. 56. As noted previously, it fails to reach the diaphragmatic surface in 17 per cent of specimens.

Resection of this segment would seem to be feasible anatomically, on account of the high origin of B^8 and the accessibility of the segment, but it would seem necessary to anticipate the numerous variations described here.

THE LATERAL BASAL SEGMENTAL BRONCHUS (B^9)

Below the origin of B^{7+8} the stem bronchus continues only a short distance (Fig. 2, *B*) before giving off the lateral segmental bronchus (B^9). On the left side, B^9 and B^{10} lie in virtually the same plane (80 per cent of specimens; Table X, I: 1) but the keel separating the two is so tilted that the orifice of B^9 lies slightly in front of B^{10}.

TABLE X. ORIGIN OF THE LATERAL BASAL BRONCHUS (B^9)

I. Arises From Basal Trunk in Common With B^{10}		90%
1. B^9 and B^{10} lie in same plane (Plate 4)	80%	
2. B^9 lies in a plane anterior to B^{10} (Plate 1, No. 52)	10%	
II. Absent as Such (i.e. displaced)		10%
1. Represented by an accessory ramus of B^7 (Fig. 7, No. 4)	7%	
2. Represented by an accessory ramus of $B^{10}a$	3%	
3. Represented by rami of B^7, B^8, and B^{10}		

Usually within a distance of 12 mm. below the keel, a prevailingly posterolateral ramus is given off in 67 per cent of specimens. This contributes to the subsuperior zone and therefore has already been described as an accessory subsuperior—BX*(9). It is the homologue of B^9a, the lateral ramus of B^9 on the right side. However, on that side B^9 rarely contributes to the subsuperior zone.

Below this ramus the main stem of B^9 continues toward the diaphragmatic surface supplying the posterolateral sector of both the costal and the diaphragmatic surfaces (Plate 2).

In 10 per cent of specimens, B^9 is absent as such, being represented by displaced rami arising as accessory branches of adjacent bronchi. In this respect it resembles the corresponding bronchus on the right side which is absent in 8 per cent of specimens. In 7 per cent it takes origin from B^7 (see BX^9 in Fig. 7, No. 4). In another 3 per cent it arises from either $B^{10}a$ or by a multiple origin from B^7, B^8, and B^{10}. In contrast to this, the accessory BX^9 of the right lower lobe always arises from B^8. In both lobes, however, the contributing bronchi are on the lateral side. As on the right, B^9 is considered to be too deeply placed to be readily resected.

THE POSTERIOR BASAL SEGMENTAL BRONCHUS (B^{10})

The posterior basal bronchus is the largest of the basal bronchi of the lower lobe and is considered by some embryologists to be a continuation of the

TABLE XI. THE POSTERIOR BASAL BRONCHUS (B^{10})

I. Mode of Bifurcation		
1. $B^{10}b$ lies in a plane deep to $B^{10}a$ (Plate 1, No. 51)		47%
2. $B^{10}b$ and $B^{10}a$ lie in the same plane (Plate 1, No. 52)		40%
3. $B^{10}b$ lies anterior to $B^{10}a$ (Plate 4, No. 60)		13%
II. The High Paravertebral Ramus of B^{10}		
1. Paravertebral ramus absent (Plate 1)		70%
2. Paravertebral ramus present		30%
(1) Arises alone from stem of B^{10} (Plate 2, No. 56)	20%	
(2) Arises in conjunction with BX*(10) (Plate 4)	10%	

stem bronchus. B^{10} courses downward, backward, and medially to terminate in lateral and medial branches ($B^{10}a$ and $B^{10}b$, respectively). Before this division occurs, one or sometimes two accessory subsuperior bronchi, BX*(10), take origin from its upper posterior surface. Also in 30 per cent of specimens one or two paravertebral branches may arise from the anterior or medial aspect of B^{10} before its bifurcation—either alone or in conjunction with an accessory subsuperior, BX* (Fig. 6, No. 23). These paravertebral branches usually supply the middle or lower portion of the paravertebral surface (Plates 2 and 4), but very rarely, if ever, supply the diaphragmatic surface.

The branch which represents the termination of the stem bronchus is $B^{10}b$ (Fig. 2, *B*). It is directed toward the inferior posteromedial angle of the lobe (Fig. 4, *B*). In origin it more often arises in a plane deep to $B^{10}a$ (47 per cent; Plate 1, No. 51), but almost as frequently it originates in the same plane (40 per cent; Plate 1, No. 52). In 13 per cent, $B^{10}b$ may lie anterior to $B^{10}a$ (Plate 4, No. 60).

The lower posterior branches of $B^{10}b$, which supply the area below the subsuperior zone, belong to the fourth level of Aeby (d_4). These rami are regularly present (Plate 1, No. 52).

As compared with the right (Fig. 4), the left posterior basal bronchus has a greater anterior extent, having moved up to the pulmonary ligament to take over the paravertebral area vacated by B^7b.

Like the lateral basal, B^{10} has too deep an origin within the substance of the lung to be considered readily resectable.

SUMMARY

An analysis of the bronchial tree of sixty left lower lobes has been presented. The prevailing pattern and its variations have been described and compared with those of the right lower lobe.

The *superior segmental bronchus* (B^6) has the same apparent mode of branching as on the right side, namely, a bifurcation, but it differs in formula—"B^6a and B^6b+c" instead of "B^6a+b and B^6c." The lower and independent origin of B^6a in the left lung seems to permit a greater paravertebral extent in that it reaches the lower third of the paravertebral surface in 57 per cent of specimens. As a consequence the superior segment caps the basal segments obliquely, whereas on the right side its orientation is prevailingly horizontal. This presents an anatomic hazard to the resection of this segment. A large

posterior branch of this ramus, B^6a2, invades the posterior sector of the subsuperior zone in 45 per cent of specimens.

The *subsuperior zone* is present in 100 per cent of specimens. It is a wedge-shaped district that is interpolated between the superior and basal segments on the costal surface of the lobe. It derives its name from an independent dorsal ramus (the d_2 of Aeby) which arises from the stem bronchus below the superior segmental bronchus. This "subsuperior proper" (B^*) occurs less frequently on the left (29 per cent) than on the right side (62 per cent). In the left lung it distributes primarily to the posterolateral sector of the costal surface but may spread laterally or posteriorly. (On the right side it distributes primarily to the posterior sector.) When it is absent, on the left, its place is taken by high posterior or posterolateral branches of the corresponding basal bronchi, $BX^*(10)$ and $BX^*(9)$. These "accessory subsuperiors" may also supplement B^* when it is present. (On the right side the zone is supplied primarily by B^* and $BX^*[10]$.) On both sides, according to Neil and others, the subsuperior zone is an important "site of localized pathological change."

The *basal bronchi* have generally been considered to be three in number—the anteromedial basal (B^{7+8}), the lateral basal (B^9), and the posterior basal (B^{10})—and to arise as a trifurcation of the lower lobe bronchus. As a result of the detailed analysis of sixty specimens it is believed that both ideas should be revised. First, the prevailing mode of branching (87 per cent) is a bifurcation, the common formula (67 per cent) being B^{7+8} and B^{9+10}. Second, it would seem necessary to give B^7 and B^8 an independent status. Whether they should be called subsegmental or segmental bronchi is of lesser consequence.

The *medial basal bronchus* (B^7) arises with B^8 from a short common stem in 87 per cent of specimens. Consequently, in contrast to its homologue on the right, its origin is anterolateral instead of anteromedial. Correspondingly its distribution shifts laterally, its terminal branches covering, in 63 per cent of specimens, the whole inferior, anterior surface of the lobe from pulmonary ligament to interlobar margin. It has never been found to extend medially beyond the pulmonary ligament, whereas on the right it regularly reaches the paravertebral surface. In 10 per cent, it arises separately from B^8 and in 3 per cent it is absent as such. On account of the accessibility of its branches and its superficial position, B^7 (as well as B^8) is considered readily resectable.

The *anterior basal bronchus* (B^8) is the most variable and defective of the basal bronchi. In 27 per cent it is either defective in origin or fails to reach the diaphragmatic surface, or both. Its lateral ramus (B^8a) may be replaced by branches arising from B^* or $BX^*(9)$ and its basal ramus (B^8b) may be replaced by accessory branches of B^9, B^7, or even the basal trunk. B^8 thus presents a sharp contrast to the anterior basal of the right lung, which is described as a most constant basal bronchus.

The *lateral basal bronchus* is much like that of the right side. It is absent as such in 10 per cent of specimens, the segment then being supplied by accessory rami of B^7, B^8, or B^{10}.

The *posterior basal bronchus* is the largest of the basal bronchi and is considered to be the continuation of the stem bronchus. It gives origin to a high

paravertebral branch in 30 per cent of specimens. Both B^9 and B^{10} originate too deeply to be considered readily resectable as individual units.

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ALVEOLAR CELL CARCINOMA OF THE LUNG (PULMONARY ADENOMATOSIS, JAGZIEKTE?)

A Multicentric Tumor of Epithelial Origin

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IT IS customary to think of a carcinoma as being a local disease, at least of having originated in a small area. It is that conception, of course, which is responsible for the prevalence of surgical attack on the condition. That the conception is correct seems to be borne out by the frequent success in obtaining permanent cures by the surgical removal of the diseased part together with the extensions of the growth from its original site. Regarding primary carcinoma of the lung the generally accepted idea, based on evidence accumulated within recent years, is that this condition not only arises in a bronchus, and is therefore more properly called bronchiogenic carcinoma, but that also like other carcinomas it arises in one spot or area. There can be no doubt that this concept is true for the overwhelming majority of cases of primary carcinoma of the lung.

Yet a few case reports have appeared describing a type of tumor, resembling a carcinoma, which seems to have arisen in the cells lining the alveoli rather than in a bronchus. Moreover, this peculiar tumor seems to have a multicentric origin and thus differs from the common and well-recognized bronchiogenic carcinoma. In this respect it has some resemblance to the primary cancer of the lung in the mouse familiar to the geneticists. Also it resembles the interesting and supposedly virus disease of South African sheep, known as jagziekte.

Several interesting questions arise in connection with this condition. If it is a true carcinoma arising in the alveoli, then the controversy about whether or not the alveoli are lined with epithelium seems apparently to be settled in the affirmative. The similarity to jagziekte raises the question of a possible virus infection in man similar to that in the sheep. Finally the question of the best surgical treatment arises.

It is because of these various considerations that the following case is presented. A special reason for reporting it is that apparently it has the longest follow-up period of any case in which the patient has been subjected to surgical treatment. The result seems to indicate that a two-year period of freedom from a manifestation of symptoms after a lobectomy gives no assurance of complete eradication of the disease, as proposed by Osserman and Neuhof[26] from their experience with a somewhat similar case.

Due to the differences in opinion as to its origin, the particular type of lung tumor characterized by the presence of malignant cells lining the alveoli and maintaining for the most part the general alveolar structure of the pulmonary parenchyma (in the absence of any demonstrable primary lesion elsewhere in the bronchial tree) has been given almost as many different names as the number of authors who have described the lesion. Despite the number of reports in the

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literature now there is still no uniformity of opinion in accepting the tumor as originating either in epithelial or mesenchymal elements, or, indeed in fact, in agreeing that it represents a tumor having a multicentric origin.

It has seemed to us, however, that the preponderance of evidence available at present allows us to consider this particular malignant pulmonary lesion as multicentric in origin and derived from endodermal elements. Consequently, it is permissible to encourage the use of the term "alveolar cell carcinoma," implying as it does the presence of epithelial cells lining the alveoli, malignant changes in which are responsible for the production of the tumor under discussion, and we shall attempt to present evidence offering a basis for this opinion. If a single name can thus be utilized in designating this specific lesion, the recognition that it is a distinct entity will be further encouraged, and clinical, pathologic, and statistical knowledge of this lung tumor gathered on a more certain footing. Future inaccurate tabulation of alveolar cell carcinoma as a form of adenocarcinoma of the lung of bronchiogenic origin can also be prevented by this means.

The historical data, incidence, signs and symptoms, and pathology were fully discussed recently by Ikeda[15] and it is not proposed to enlarge upon these phases of the subject. In brief, the tumor has been reported to occur in two forms, a nodular variety and a diffuse type in which confluence of the tumorous areas suggests a pneumonic picture in the gross. The lesions may be restricted to one lobe of a single lung or involve all lobes of both lungs simultaneously, and they occur within the lung parenchyma or appear on the pleural surface. On gross examination the nodules are commonly discrete with good definition from the surrounding tissue, but they may be irregular in outline or coalesce, this coalescence probably representing the basis for the diffuse form of the disease. The bronchi are not involved except as a result of direct invasion by one of the nodular areas, but metastases do take place to the regional lymph nodes, and occasionally to distant organs. The presence of the tumor in the vicinity of a bronchus may cause atelectatic changes and later inflammatory processes in the adjacent and peripherally situated parenchyma.

Microscopically, the tumor is easily recognized by its characteristic morphology, the malignant cells lining the alveoli, and the general structure of the parenchyma being maintained in most areas. The individual cells tend to be polygonal in shape with rather dark-staining, abundant acidophilic cytoplasm and may occur in multiple layers or in papillary projections within the alveoli, which are often completely filled by these formations of malignant cells. The nuclei are somewhat irregular, often hyperchromatic, and mitoses may be seen in keeping with the obvious neoplastic nature of the cells. An outstanding feature is the general lack of invasion of the supporting framework of the lungs, although occasionally a lymphatic vessel may be seen filled with malignant cells (as might be expected from the occurrence of lymph gland metastases) and in a few of the more malignant cases, the cells are found growing wildly in masses and cords among a loose fibrous tissue stroma. The fundamental feature in the recognition of the tumor is, of course, the very definite reproduction of alveolar structure. It is customary to find many frankly malignant areas that are completely sepa-

rated as far as gross and histologic evidence is concerned, and occasionally other discrete areas are discovered in which single alveoli may show early hyperplasia of the lining cells of the alveolus, suggesting the beginning of still another focus of the tumor.

In the discussion to follow, this tumor is considered under two headings in accordance with the conflicting views held concerning its origin, (a) the multicentric nature of alveolar cell carcinoma of the lung, and (b) the epithelial origin of alveolar cell carcinoma of the lung.

THE MULTICENTRIC NATURE OF ALVEOLAR CELL CARCINOMA OF THE LUNG

Multiple primary malignancies are no longer the rarity that they were once considered to be, but it is still difficult to accept all the cases reported as actual examples of this condition for the criteria followed in assessing the cases are not always clear. However, the following studies, listed in Table I, seem well substantiated and the figures are included to illustrate the incidence of multiple primary malignancies generally without specific reference to the lung.

TABLE I

	NUMBER OF AUTOPSIES IN MALIGNANT DISEASE	CASES OF MULTIPLE PRIMARY MALIGNANCY	PERCENTAGE OF MULTIPLE MALIGNANCY
Owen, 1921	3,000	143	4.8
Orr, 1930			
(1) Personal cases	1,046	8	0.77
(2) Collected data	9,107	76	0.83
Hanlon, 1931	710	18	2.5
Warren and Gates, 1932	1,078	–	3.7
Schreiner and Wehr, 1934	11,212	307	2.7

The skin and intestinal tract are most commonly affected, with the female breasts and genital organs of both sexes involved with greatest frequency in paired structures. The rarity with which the lung is affected is illustrated in a recent study by Slaughter[29] of 1,868 collected cases of multiple primary malignancy in which he found 749 instances (40 per cent) in the same organs or systems, but not a single case in the lung, and 269 cases (14.4 per cent) in paired organs with only 3 reports of lung involvement. In addition, carcinoma of the lung occurred relatively uncommonly also in association with primary carcinomas affecting organs of other systems, there being in this series only 34 reports of such an association, representing an incidence of only 1.8 per cent as compared to 7 per cent for the stomach, 6 per cent for the prostate, and 4 per cent for the kidney.

Billroth's[6] original view (expressed in 1889) that three conditions must be satisfied before one can consider such lesions as examples of true multiple primary malignancy is now universally considered too exacting. To rule out lymphogenous and hematogenous metastases, extension by continuity or contact, and implantation or transplantation, he suggested that: (a) Each carcinoma must have a different histologic structure, (b) the histogenesis of each carcinoma from a different parent epithelium must be proved, and (c) each carcinoma must form its own metastases.

However, in certain types of multiple carcinomas, generally accepted as primary in origin, these conditions are certainly not fulfilled as, for example,

in the case of the occurrence in multiple form of basal cell carcinoma of the skin which rarely metastasizes; for this reason Goetze[11] suggested the necessary criteria might be the following: (a) The macroscopic and microscopic appearance of the tumors must be that of the usual carcinomas of the organs involved, (b) exclusion of metastases must be certain, and (c) diagnosis may be confirmed by the character of the metastases in each case.

If one now accepts on this basis that such lesions as multiple skin carcinomas and multiple malignant polypi of the colon are true multiple primary malignancies, it would seem possible to accept multiple areas of malignancy in the lung as representing multicentric neoplasia, provided that it was possible to satisfy, by meticulous pathologic examination, conditions such as those to be outlined here, which were originally described by Cassili and White[7] in deciding that a case which they reported was in fact an example of multiple lung tumors arising independently from the alveolar lining cells.

1. No primary tumor could be found elsewhere in the body.
2. No primary tumor could be demonstrated in the bronchial tree. In this connection Weller[33] has stated that one must always search until the primary lesion is actually found, because he feels that all such multiple lesions are the result of lymphatic spread.
3. The tumor must be multicentric with areas of uninvolved parenchyma separating the malignant nodules.
4. The neoplastic cellular process must be confined to the alveolar sacs.
5. The interstitial framework of the lungs, except for the lymphatic vessels, must be generally free of the process.
6. The anatomy and histology of the nodules must be unlike any other primary lung process.

   (The fact that all lobes of the lungs were affected in their case seems of relatively little importance.)

Ever since Müller[21] divided the cases in which multiple primary malignancy was present into three groups (1) involving the same system (for example, skin and intestinal tract), (2) involving symmetrical paired organs (for example, breasts, ovary, testis, kidney, lung) and (3) involving distant organs of separate systems, it has been most difficult to satisfy the necessary criteria in the second group (including as it does the breast and genital organs); and it has also been customary to exclude from consideration (as do Warren and Gates[32]) cases of malignancy which is frequently multicentric in origin affecting a single visceral organ, such as carcinoma of the liver, although there seems little doubt that the separate lesions in this case arise independently. As a result of this latter exclusion, similar lesions of the lungs, such as those under consideration in this paper, have also commonly not been included in studies of multiple primary malignancy.

Although multiple lesions of the skin and those occurring in widely separated colonic polypi are now generally regarded as distinct primary changes, it is more difficult to rule out a metastatic basis in the case of paired organs such as the breast and the ovary, where multiple malignancies are next most commonly seen. As mentioned previously, reports in the literature venturing to call these

lesions primary in type are often open to criticism. In fact Warren and Gates[32] also exclude most of these cases from their survey.

It is perhaps more difficult still to be certain that multiple lesions in a single lung can be considered to have arisen independently when spread may have occurred along alveolar walls, by the readily available vascular or lymphatic channels or by the intraluminal dissemination of cells with the air motion of ordinary or forced respiration. Nonetheless, sufficient reports have now appeared in which the histologic features are essentially the same regardless of the fact that the change may appear in discrete and widely separated areas or show a tendency to become confluent that it seems unlikely that spread by any of these means could have been responsible, particularly when the conditions outlined according to Casilli and White[7] are satisfied.

The observation that the experimentally produced lesions of alveolar hyperplasia, adenomatosis, and carcinomatosis to be mentioned later are commonly multiple in occurrence and that many of these changes appear simultaneously lends further credence to the view that similar changes occurring spontaneously in the human being are multicentric. In addition, Ikeda[15] stressed the point that in one of the cases he reported each of the nodules actually represented a well-developed intra-alveolar papilloma with a definite central fibrous core and he felt that this further observation also favored the theory of multicentric origin of the tumor.

In summary, then, it becomes apparent that this specific entity which has probably often been labeled an adenocarcinoma in the past is being reported with increasing frequency (for example, Adler[1] 20 cases in 1912, Neuberger and Geever[22] 25 cases in 1942, Ikeda[15] 4 cases in 1945) as its distinctive histologic appearance is accepted, and the typically multiple arrangement of neoplastic areas coupled with the failure to demonstrate a hidden primary tumor permit general acceptance of the view that it has a multicentric origin.

THE EPITHELIAL ORIGIN OF ALVEOLAR CELL CARCINOMA OF THE LUNG

Just as there has been hesitation in accepting the multicentric origin of pulmonary tumors having this distinctive histologic appearance, so also has there been lack of general agreement concerning the presence or absence of a layer of cells lining the alveoli from which this neoplasm might arise. Furthermore, disagreement has been apparent in deciding whether this layer of cells, if it is actually present, develops from endodermal or mesenchymal elements and, consequently, whether it is proper to name the tumor a carcinoma at all.

It is undoubtedly difficult, if not impossible, to demonstrate a lining layer of cells in the alveoli of normal lung tissue, but as Miller[20] points out in his monograph, it is often necessary for the pathologist to turn to normal tissues when in doubt as to the correct designation of a certain cell or group of cells, and in a similar manner the normal histologist may be obliged to inspect pathologic material in order to obtain better insight into the structures under observation. In the case of the lung, he has shown beautifully the presence of a continuous layer of lining cells in the alveoli when they are rendered visible by being separated from the underlying framework of connective tissue and vessels by (a) the pouring out of a serous exudate behind them in a pneumonia or (b)

the mechanical edema of mitral stenosis with insufficiency of the valve. In these cases the lining appears as a layer of flattened epithelium in a continuous sheet, the nucleus bulging out in the thinned-out portion of the cell.

Miller[20] also pointed out that the presence of "pores" in the alveolar walls may be explained on the basis of pathologic changes occurring when the epithelium is pushed off in this manner and that they are not necessarily normally present. When the epithelium is pushed off from diametrically opposed sides of an alveolar wall, the serum poured out from the capillaries then finds an exit into adjoining alveoli and with the conversion of fibrinogen into fibrin the characteristic strands of fibrin passing through the alveolar wall are formed (see Macklin).[17] In these cases epithelial cells may be seen attached to the strands of fibrin as they leave the alveolar wall. Macklin[18] demonstrated residual epithelial cells on the pulmonary alveolar walls in various mammals, and he believes it is possible that primary carcinoma of the lung can originate from these cells in man.

Nonetheless, it has been repeatedly observed that cells may be seen lining the alveoli in certain diseased conditions such as chronic passive congestion, lipoid pneumonia, chronic interstitial pneumonia, and other chronic inflammatory processes such as empyema, abscess, bronchiectasis, and tuberculosis. The process appears to be one of hyperplasia of the lining cells and has been termed "epithelization" of the alveoli.

Similar changes which may progress to adenomatous formations and frank malignancy can be produced experimentally by the use of certain chemical carcinogenic compounds. Andervont[2] was able to show for the first time in the lung that carcinogens do not act only locally when he produced primary lung tumors in a certain pure strain of mice by the subcutaneous injection of dibenzanthracene, usually without the occurrence of subcutaneous tumors. Although this strain of mice had a high incidence of spontaneous lung tumors, the induced growth appeared much earlier and it was postulated that following absorption of the carcinogen from the subcutaneous tissue it was carried to the lung where it accelerated the normally occurring carcinogenesis. Lung tumors could, of course, also be produced by the implantation of dibenzanthracene directly into the lungs of this mouse strain.

It is interesting to note that Andervont[3] also bred male and female mice of high pulmonary tumor strain to females and males, respectively, of low pulmonary tumor strain, where all attempts at the induction of lung tumors by dibenzanthracene had failed, and following this females of the first hybrid generation were then mated to their brothers to produce the second hybrid generation. The presence of tumors in a high percentage of both the first (90 per cent) and the second (75 per cent) hybrid generation shows that the susceptibility of the lung to tumor formation induced by the subcutaneous injection of a lard-dibenzanthracene solution is inherited in a dominant manner and is clearly specific for that particular organ.

Multiple adenomatous tumors originating along the alveolar walls have been produced experimentally in mice by Grady and Stewart,[12] and spontaneous multiple tumors of the same type, also in mice, have been observed by Slye, Holmes, and Wells.[30] These adenomatous lesions resemble those described by Cowdry[8] in

South African sheep, a disease known as jagziekte and probably caused by a virus, the counterpart of which has been reported in human beings under the name of adenomatosis by Bell.[5]

We then have definite cellular changes occurring in the alveolar walls in response to many of the factors that have been shown by those working in cancer research to be potentially responsible for tumor formation in general. The factors mentioned have been (a) chronic irritation (chronic inflammations in human beings), (b) chemical carcinogenic compounds (in mice), (c) heredity (in mice), (d) tissue susceptibility (in mice), and finally (e) viruses (in sheep).

Surely, if such responses (hyperplasia or "epithelization" adenomatosis and malignant degeneration) can be regularly induced in the alveolar walls by such varying factors, it would seem that the presence of a cellular lining of the alveoli can no longer be denied, particularly in view of Miller's[20] masterful demonstration of its existence.

However, even if this reasoning is accepted, the question concerning the exact developmental nature of a lesion arising from these cells has still not been answered, for we have yet to decide whether they are of epithelial origin or have a mesenchymal background. No definite answer to this problem seems possible at the present but several factors are highly suggestive.

It is generally accepted by embryologists that the lung develops as a bud of endoderm from the foregut, which branches out into the surrounding mesenchyme as the lungs project into the thoracic celom. On this basis it would seem reasonable to expect that the sacculations (or alveoli) at the end of the branching tree would retain a layer of cells derived from the endodermal bud from which they developed. However, as Barnard and Day[4] pointed out, it has been noted that this cuboidal lining which is admittedly present in early embryonic life seems to disappear from about the fifth month onward and has not been seen to reappear at a later date. Quite possibly in view of Miller's[20] demonstration that a lining does exist, restudy of this problem with more modern techniques, as for example an artificial duplication of the increased venous pressure found in mitral stenosis, might show a similar lining of flattened cells that was not apparent before.

Those who believe that this lining layer does actually disappear theorize that the changes of hyperplasia, adenomatosis, or frank carcinomatosis, as described previously, occur as a result either of hyperplasia of the so-called "septal cells" (Maximow and Bloom)[19] which have a perivascular distribution and are probably mesenchymal in origin, or of extension into the alveoli of cells lining the bronchioles, the epithelial origin of which is uniformly accepted. In the first instance these mesenchymal septal cells may, as Neuberger[23] suggested, gradually form a continuous lining which becomes clearly demarcated from the adjacent tissues, but the absence of reticulum fibrils and the cellular appearance so closely resembling the usual adenomas or carcinomas make this theory appear improbable. It is only fitting to point out, however, that this latter objection is overcome if one accepts the views of Oppenheimer[24] (quoted by Neuberger and Geever) who does not believe that germ layers are specific and believes them capable of undergoing transformation from one type to another under different environmental conditions.

In supporting the second theory Herbut[14] favored the origin of the alveolar cells from the basal cells of the bronchioles since the fact that there is a continuation of these basal cells into the alveolar lining in some cases of bronchiectasis shows that such invasion is possible. Sweany[31] and Jaffe[16] also believe that these basal cells are multipotential and can develop into any type of cell in the bronchial tree including the so-called "alveolar cell," but actually in the literature there does not seem to be any irrefutable histologic evidence that extension of these cells has occurred into the alveoli in the lesions found in this particular type of pulmonary neoplasm.

In this regard Frissel and Knox[10] felt that the alveolar pattern of cellular arrangement merely represents an attempt on the part of the tumor cells to maintain proximity to the available vascular supply in the walls of air sacs and on this basis they felt justified in classifying the lesion as an adenocarcinoma of bronchiogenic origin.

Although it is impossible with our present state of proved knowledge to discard these other theories completely, we believe that there is a sufficient bulk of evidence favoring the presence of a definite cellular alveolar lining to accept its presence and also to consider the cells to be of epithelial rather than mesenchymal origin. Therefore, the term "alveolar cell carcinoma" would appear to be the one most suitable for the future designation of this specific pulmonary neoplasm.

CASE REPORT

The patient, E. L. W., a 35-year-old used car dealer, was admitted to Barnes Hospital on June 1, 1943, at which time he stated that he had suffered from a chronic cough for the past eight to twelve months, which had become productive in the six months prior to admission. At this time he was coughing up approximately one-half cup of foul-smelling, greenish yellow sputum each day, and although there had been no associated pain, voice changes, general weakness, or weight loss, he stated that he had noted a small amount of blood streaking of the sputum ten days before admission. This was the only occasion on which hemoptysis had been present.

There had been some dyspnea on exertion, associated with palpitation and vertigo, and these "dizzy spells" had become sufficiently severe that the patient had consulted a physician concerning them about six months before he was admitted and had been treated for "heart trouble" and "allergy" without any improvement. One month before admission he had been seen by a chest specialist who ordered a chest plate and subsequently referred the patient directly to a hospital.

The past history apart from some mild chronic sinusitis of many years' standing and a "touch of dropsy" did not appear to be significant, nor did the family history or the general functional inquiry. He did not smoke.

Physical examination at the time of admission revealed a few large moist râles at the lung bases posteriorly, with some questionable diminution in the audibility of the breath sounds over the right lung field. The temperature, pulse, respiration, blood pressure, and laboratory findings were within normal limits and the sputum was negative for tubercle bacilli or fungi. There were no other significant findings on physical examination.

A flat plate of the chest showed an infiltrative process in the right lower lung field (Fig. 1) which was nonspecific, and a lipiodol bronchogram showed all the bronchi on the left side as well as those to the right upper and middle lobes to be normal. Only a portion of the right lower lobe bronchus was filled, although several flecks of opaque material were visualized overlying the large area of infiltration.

Diagnostic bronchoscopy was similarly of little help. The trachea contained a little pus but the carina was thin and normal in appearance and the only noteworthy finding in any of the bronchi visualized was a slight bulging in one of the medial divisions of the right lower lobe bronchus from which a biopsy was taken. The pathologic report on the specimen, however, showed no areas suspicious of malignancy and was labeled as chronic inflammation. The material consisted of a polypoid mass covered on all sides with epithelium that occasionally seemed to be undergoing squamous metaplasia. There was a core of fibrous tissue with many blood vessels and a considerable inflammatory processs throughout although most of the cells were lymphocytes.

It was felt that an exploratory thoracotomy should be performed and this was done (by E. A. G.) on June 8, 1943.

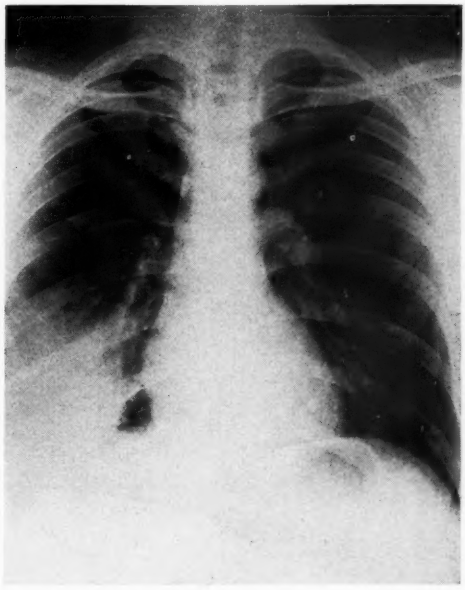

Fig. 1.—Roentgenogram of chest June 2, 1943, just prior to first operation. Homogeneous poorly delineated shadow in right lower chest. Upper lung field clear.

Operation.—On opening the chest there was no evidence of either pleural effusion or adhesions, but there was a mass of fairly firm tissue involving the greater portion of the right lower lobe and extending to the periphery where it caused a very marked dimpling of the visceral pleural somewhat suggesting carcinoma. However, it was felt that the consistency was not sufficiently firm to substantiate this suggestion and it was finally decided that the lesion was inflammatory in nature and consequently a lobectomy was performed. Ordinarily a frozen section would have been made at the time of operation, but the limited facilities for carrying it out because of war conditions made the procedure impossible.

A considerable amount of bloodstained fluid collected in the immediate postoperative period, and as closure had been effected without drainage, it was necessary to perform several thoracenteses to prevent a shift of the mediastinal structures. No evidence of infection appeared, however, and thereafter the postoperative course was uneventful and recovery satisfactory.

Pathologic Report.—The gross appearance was much as described previously with a depressed puckered looking area overlying the actual lesion, which on sectioning was found to comprise a well-encapsulated area of edematous and probably infected tissue. At one edge

of this area was a small cavity containing some necrotic tissue, and surrounding this cavity the induration was sufficient to re-arouse the suspicion of carcinoma. It also suggested an organizing pneumonia (Fig. 2).

Fig. 2.—Gross specimen of right lower lobe removed June 8, 1943. Note gray-white area which suggests pneumonic process surrounding zone of cavitation. No lymph nodes involved.

Microscopically the tendency to reproduce the alveolar structure was by far the predominating feature of the lesion. Several well-defined areas of lung tissue showed almost complete replacement of the alveolar linings by hyperplastic epithelium comprised of polygonal cells having abundant acidophilic cytoplasm and dark-staining nuclei (Figs. 3 and 4). At the periphery of the lesion there were occasional isolated groups of alveoli in which the epithelium looked hyperplastic with increase in the cellularity, the cells tending to grow in small papillomatous formations projecting into the lumen of the alveoli (Fig. 5). In still other areas the tendency to the reproduction of alveolar structure was not seen, the cells growing wildly in clumps and cords in a loose fibrous tissue stroma. In these zones the malignant cells showed marked variation in size, shape, and staining reaction, and mitotic figures were fairly frequent. The remaining lung parenchyma showed a peculiar lobular distribution of edema and there was a marked chronic inflammatory reaction surrounding all the

larger bronchi and bronchioles. There was no evidence either at operation or pathologically of involvement of the regional lymph nodes.

Although there was obviously a great deal of secondary infection and inflammation in the lobe it was recognized at the time by Dr. Nathan Womack that a carcinomatous process appeared to be arising simultaneously in several different regions. Also he called attention to the similarity of this case to jagziekte. Because of this fact and the lack of any evidence of regional metastases, it was decided unwise to consider completion of the pneumonectomy due to the probability that similar areas of involvement were present in the left lung. Perhaps this was an error in judgment in view of the subsequent history.

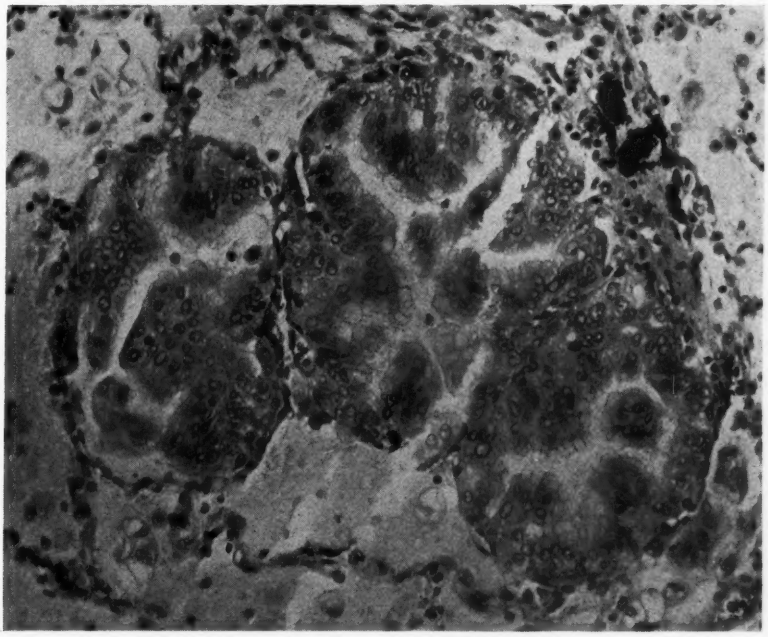

Fig. 3.—Characteristic nodule of well-differentiated tumor with preservation of alveolar pattern surrounded by normal lung parenchyma, first operation. (Photomicrograph ×420.)

Subsequent Course.—The patient made a satisfactory recovery and was well until the fall of 1946. He saw Dr. A. S. Ashmore of Wichita, Kan., in October, 1946, and complained at that time of a productive cough. An x-ray of the chest showed a lesion overlying the diaphragm measuring 2½ inches in diameter. He refused treatment at that time. He began to lose weight and developed pain in the right upper thorax and shoulder girdle region which interfered with the function of the right arm. On July 17, 1947, bronchoscopy was negative and the lipiodol examination was unsatisfactory, but the x-ray demonstrated a dense shadow obscuring the right lower lung field (Fig. 6). In December, 1947, exploration was done at Wichita by Dr. Ashmore and the remaining portion of the right lung resected. At the time of the exploration, tumor resembling "caviar" or "raspberry jelly" was seen growing over the chest wall where it had invaded over an area of 4 inches in diameter. The mediastinal lymph nodes were apparently negative. He made another uneventful recovery from this operation but because of the evidence of invasion of local tissues, he was given intensive postoperative irradiation therapy (7,900 r. through four portals). He was last seen in September, 1948. Bronchoscopy was negative and there were no tumor cells found in the smears from the bronchus. The opposite lung was clear to radiographic examination. He was in

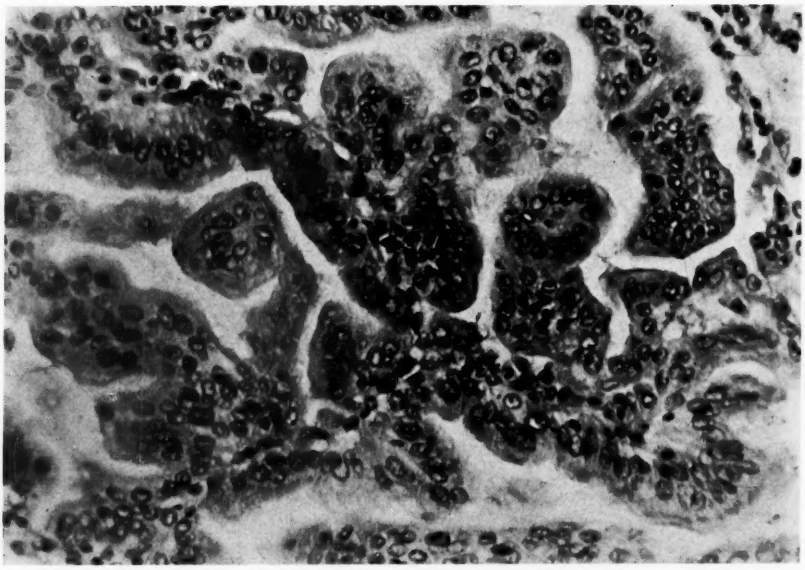

Fig. 4.—Detailed view of nodule of tumor demonstrating its well-differentiated character, first operation. (Photomicrograph ×420.)

Fig. 5.—Cluster of tumor cells suggesting origin from alveolar epithelium, first operation. (Photomicrograph ×650.)

excellent clinical condition. The microscopic pattern of the tumor found at the second operation was very similar to that seen at the first operation (Fig. 7).*

DISCUSSION OF CASE

This patient has a known duration of his disease of at least six years. The initial lobectomy resulted in freedom from symptoms for about four years. At the time of the second operation the microscopic appearance of the tumor had not changed (Fig. 7). However, there was evidence of local invasion of the chest wall. The regional lymph nodes have remained free from disease. The opposite lung radiographically has shown no change but it is entirely possible that already in this lung there may be the early changes of hyperplasia (epithelization) or benign adenomatosis that are presumably of a precancerous nature.

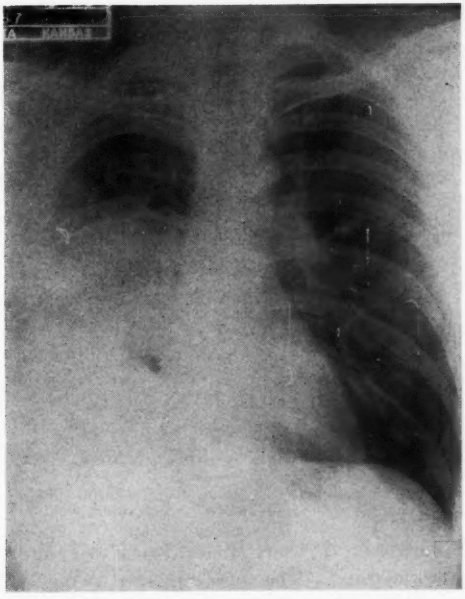

Fig. 6.—Roentgenogram of chest July 8, 1947, just prior to second operation. Dense shadow in right lower chest, high diaphragm, thickened pleura. Left lung clear.

The surgical treatment of this case has resulted at least in very worth-while palliation. The peculiar nature of the tumor, although probably malignant, and the absence of any known surgical experience with it prior to our operation in 1943, made the necessity of total pneumonectomy seem questionable at that time. Perhaps, however, further experience will show that the total removal of the lung is indicated as a palliative procedure in spite of the existing possibility of lesions in the other lung. This case would seem to suggest that. This patient is apparently the third treated surgically and the only one with a long follow-up (Drymalski[9] and Wood and Pierson[34]). Wood's patient had a right lower lobectomy for what was thought to be an inflammatory lesion. A roentgenogram six weeks after operation appeared to be clear of disease. The patient died six

*We are indebted to Dr. Ashmore for the history of the subsequent course, the x-ray film, and the microscopic sections from the second operation.

months later of carcinoma of the uterus. At post-mortem examination multiple foci of pulmonary adenomatosis were found in all remaining lobes. As Wood's case is very similar to the one reported here in both its clinical course and pathology, it lends further support to the concept of multiple foci of origin. His case also suggests that the case reported here may very well have other foci of tumor in the remaining left lung. Wood recognized the similarity of his case to jagziekte. No doubt the lobar distribution of the signs and symptoms of some of these cases is due to the strategic location of nodules of tumor close to a bronchus. This results in infection distal to the block and these changes give a clinical pic-

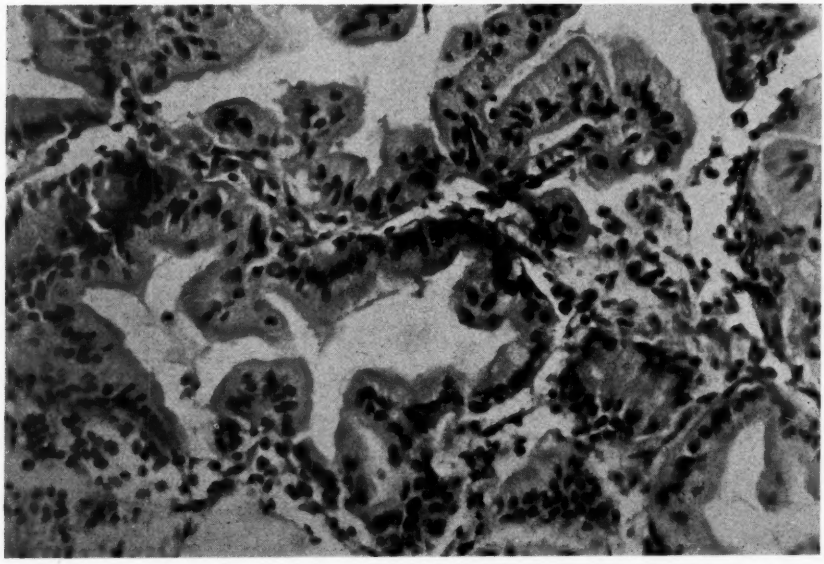

Fig. 7.—Persistent tumor in upper lobe. Note well-differentiated character and similarity to original tumor, second operation. (Photomicrograph ×60.)

ture suggesting inflammation. If there is no surgical interference the patient dies of inflammatory complications. The case reported demonstrates the value of surgical resection in the presence of such localized inflammatory complications.

SUMMARY

A brief review has been presented dealing with a specific type of pulmonary neoplasm which is characterized by a reproduction of the alveolar structure of the lung. The evidence is discussed which favors recognition of the multicentric nature of the lesion and seems to indicate that it may quite properly be called an "alveolar cell carcinoma." An illustrative typical case is reported which demonstrates the slow growth of the neoplasm and the palliation which may result from the removal of the diseased tissue, although the manifestations of the disease may reappear several years later in a different part of the lungs, as in this case. Also, attention is called to the similarity of this case to jagziekte, the South African disease of sheep.*

*The pathology of this case has been reviewed by Dr. Lauren V. Ackerman of the Section of Surgical Pathology, Washington University School of Medicine, St. Louis, Mo.

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ECTOPIA BRONCHI

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DEVELOPMENTAL abnormalities of the tracheobronchial tree and of the pulmonary parenchyma are rarely found in life or at necropsy. Even after careful clinical scrutiny one commonly confuses such abnormalities with other pulmonary diseases, or else arrives at a diagnostic impasse. We are principally interested here with ectopia of the bronchi to the left upper lobe and with the resultant varied parenchymal changes in the left lung.

EMBRYOLOGY

Arey[1] in discussing the embryology of the lung stated that in the 3 mm. human embryo the lung first appears as a groovelike evagination on the ventral surface of the esophagus. The enlarged caudad end of this evagination gives origin to two small lung buds. At a later stage, the predestined anlagen of the esophagus and trachea become separated by a constriction. This constriction is separated at the cephalic end by the larynx which is demonstrable at the end of the fifth week in the embryo. Hollow evaginations of the lung buds extend into the surrounding mesenchyme, enlarge rapidly, and branch to produce a treelike tubular system. The surrounding mesenchyme by the end of the seventh week gives rise to muscle fibers, cartilaginous rings, and probably to the lining cells of the alveoli.

Ekehorn[2] and Flint[3] related that the lower, blind end of the primordial trachea becomes fixed, divides, and then projects into each thorax (stem bronchi) where, by a process of arborization and downward growth, the bronchial tree is developed. Heiss[4] believed that a process of infolding and septum formation are the developmental mechanics responsible for the progressive differentiation of the lung. He rejected the term and conception of stem bronchi. For him, stem bronchi are the periphery of the lung from which the connecting and terminal structures of the lung parenchyma are formed by a continuous infolding.

Ferguson and Neuhauser[5] added that as a result of the sequence of embryologic events "complete absence of the lung, with no lung bud or main stem bronchus whatever, indicates a developmental defect which has occurred very early in fetal life."

Gruenfeld and Gray[6] speculated that developmental anomalies of the lung are caused by "phylogenetic newness of the organ" and by the "enormous growth it must undergo in the embryo before its functional form is achieved." They first identify the division of the left stem bronchus into its two main bronchi in the 7 mm. length human embryo. It is interesting that in their review of lung malformations there is not included an example of the type here presented.

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Schneider[7] classified aplastic lungs according to the presence or absence of a rudimentary bronchus or of an aplastic lung. He further differentiated such aplasia into high and low degrees depending on the absence in whole or in part of the bronchial or pulmonary element.

Jordan[8] in his detailed classification of pulmonary anomalies would include the case herein reported as "agenesis of bronchus—to a normal lobe."

Caffey[9] in describing the congenital defects associated with total aplasia of the lung included ". . . . diaphragmatic defects, cardiac and renal malformations, skeletal abnormalities, and absence of the vagus nerve." He considered that the high frequency of association of pulmonary aplasia with hemivertebra justifies considering the combination as a "syndrome." He presented a case showing bronchographic evidence of absence of the eparterial bronchus in association with an hemivertebra of the eleventh thoracic vertebra. Peeler and Phillips[10] cited the finding in a 2-month-old infant of a hypoplastic bronchus to the left upper lobe; however, there was no histologic examination. Cassinelli[11] and Nelson[12] each discussed a similar case which on microscopic examination was found to have a marked diminution in the number of cartilaginous rings. Morelli[13] discussed the possibility that an aberrant pulmonary artery might produce this latter aberration. He, too, cited a case in which the bronchus to the left upper lobe was small and lacking in cartilaginous rings and in total cartilage in the rings present. Diagnosis was made on his patient at 1 month of age, and substantiated at necropsy at 4 months of age. He indicted the aplasia of bronchus and bronchial cartilage as the agent precipitating the emphysema.

In concluding this review of the literature on the embryology of the trachea, bronchi, and lungs, we feel with Harris and Lewis[14] that knowledge of the comparative anatomy and embryology of the bronchial system has not kept pace with thoracic surgery. This lack of information is reflected in the difficult recognition of such lesions.

CASE REPORT

A white boy, aged 9 years, was admitted* April 23, 1947, to Children's Hospital, Akron, Ohio. The child's physical and mental growth and activities were not unusual until 1945, when for the first time he was noted to tire readily and to become dyspneic upon exertion. A physician made the diagnosis of dextracardia and cautioned the parents to limit the child's physical activities; as a result of this the child became acutely aware of his heart and selectively limited his activities to his own favorite sports. During the next two years his only complaint was of shortness of breath; there was no cough, expectoration, chest pain, or known evening hyperthermia.

Physical examination on admission revealed a thin, 9-year-old, intelligent, cooperative white male child in whom positive findings were limited to the thorax. The thoracic cage was asthenic in type with a widening of the intercostal spaces on the right and a narrowing and relative immobility of those on the left; there was a slight list of the left shoulder. The heart rate was 110 with a normal rhythm; the left border of cardiac dullness was retrosternal, the right was at the right nipple. On the left side there was increased resonance and diminished breath sounds and voice conduction. On the right side breath sounds were normal. There was a mild secondary anemia. Intermittent afternoon fever as high as 100.4°

*On the medical service of Dr. Noah Miller.

Fig. 1. Fig. 2.

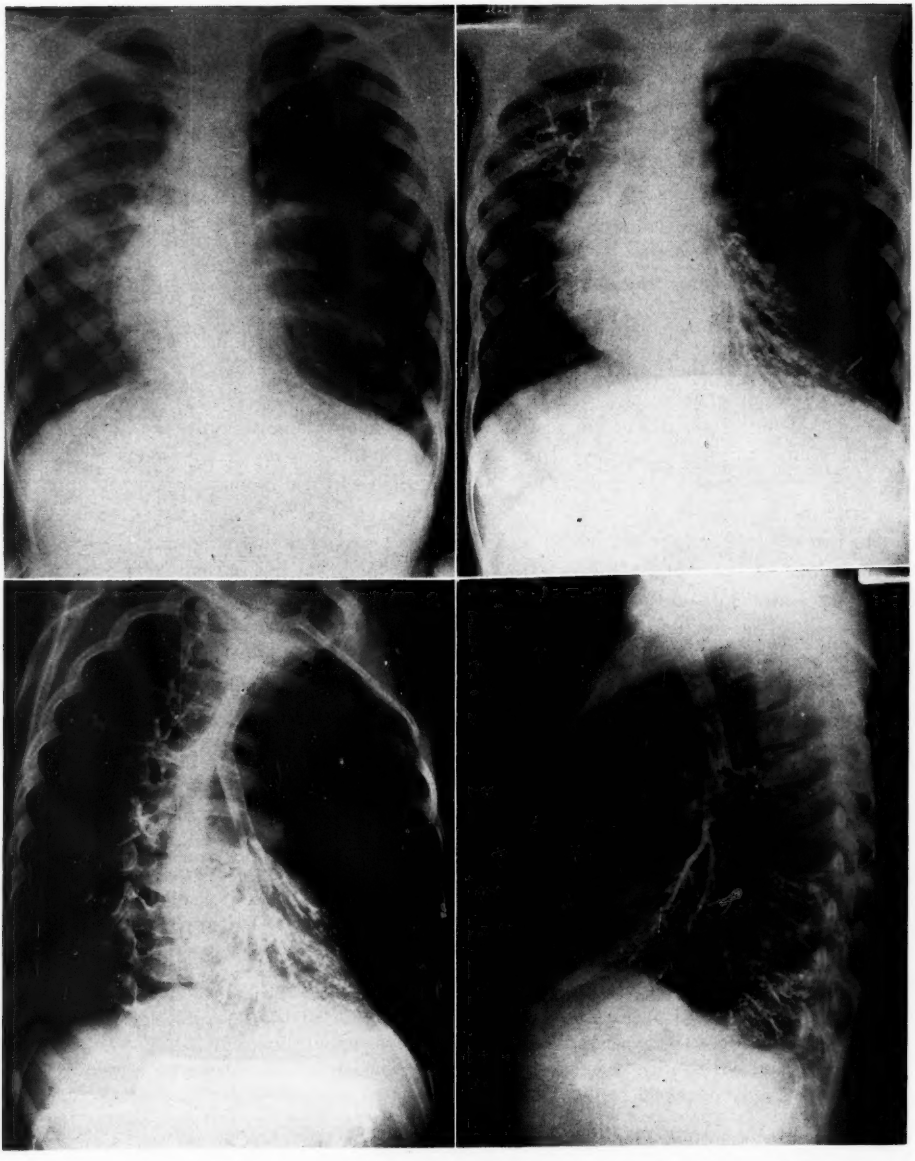

Fig. 3. Fig. 4.

Fig. 1.—Anteroposterior roentgenogram showing cardiac shift, radiolucent upper left chest, and small paramediastinal cyst.

Fig. 2.—Anteroposterior bronchogram showing concentration of bronchi in lower thorax.

Fig. 3.—Left postero-oblique bronchogram showing upper lobe bronchi in detail.

Fig. 4.—Left lateral bronchogram showing posterosuperior upper lobe bronchi extending into emphysematous mass.

F. was noted. Intrathoracic pressure readings in the second left interspace were: on inhalation, plus 2 cm. water pressure, on exhalation, plus 6½ cm. After aspiration of 200 c.c. of air the pressure readings were: on inhalation, minus 10 cm. water pressure, on exhalation, minus 4 cm. The patient coughed vigorously twice, and following this the readings returned to the pre-aspiration levels.

Anteroposterior x-ray view revealed (Fig. 1) a shift of the mediastinum to the right with a semblance of a dextracardia. A few fine pulmonary markings appeared to radiate from the left hilum into the superior and lateral aspect of the left lung area; these markings radiated from an area 3 cm. in diameter of increased density located just to the left of the hilus. A concentration of pulmonary markings in the lower fifth of the left thorax suggested a compression type of atelectasis. Bronchograms (Figs. 2, 3, and 4) revealed a concentration of an entire normal left bronchial tree in the atelectatic lower lung field, and a composite bronchodiagram, left lateral view (Fig. 5), identifies each bronchus according to the systems of Boyden[15] and Jackson and Huber.[16] In the anteroposterior (Fig. 2)

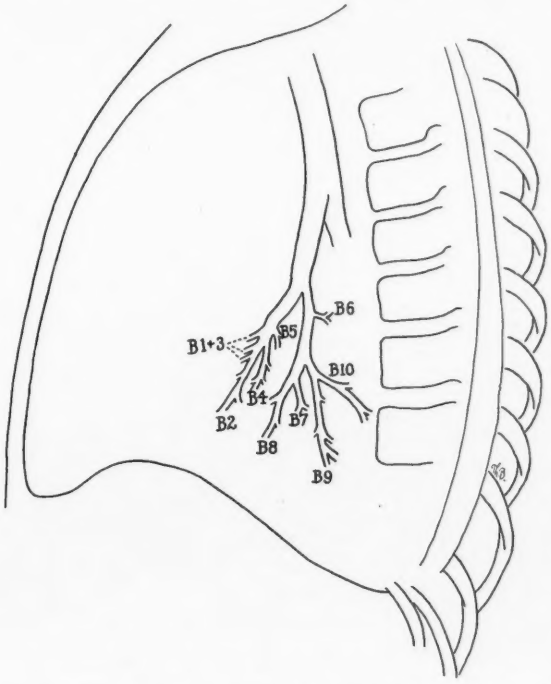

Fig. 5.—Composite bronchodiagram, left lateral, developed from Fig. 4, with identification of bronchi according to the system of Boyden,[15] and correlation (below) to the system of Jackson and Huber.[16]

UPPER LOBE

BOYDEN	JACKSON AND HUBER
B 1 plus 3	Apical-posterior
B 2	Anterior
B 4	Superior (lingular)
B 5	Inferior (lingular)
LOWER LOBE	
B 6	Superior
B 7)	Anterior-medial basal
B 8)	
B 9	Lateral basal
B 10	Posterior basal

and left lateral (Fig. 4) views there are several fine branches extending cephalad from the first 2 cm. of the upper of the two stem branches of the left main bronchus. These left upper lobe branches are identified (Fig. 5) as Bronchi 1 and 3 by the system of Boyden[15] and Scannell[17] or the posterosuperior segmental bronchi by Jackson and Huber.[16] It was subsequently determined that these bronchi were the only sources of air into the emphysematous upper lobe. The normal complement of upper lobe bronchi readily can be identified in the left posterior oblique view (Fig. 3), and the two bronchi to the lingula are seen to advantage in the lateral view (Fig. 4) to arise as separate branches from the lobe bronchus.

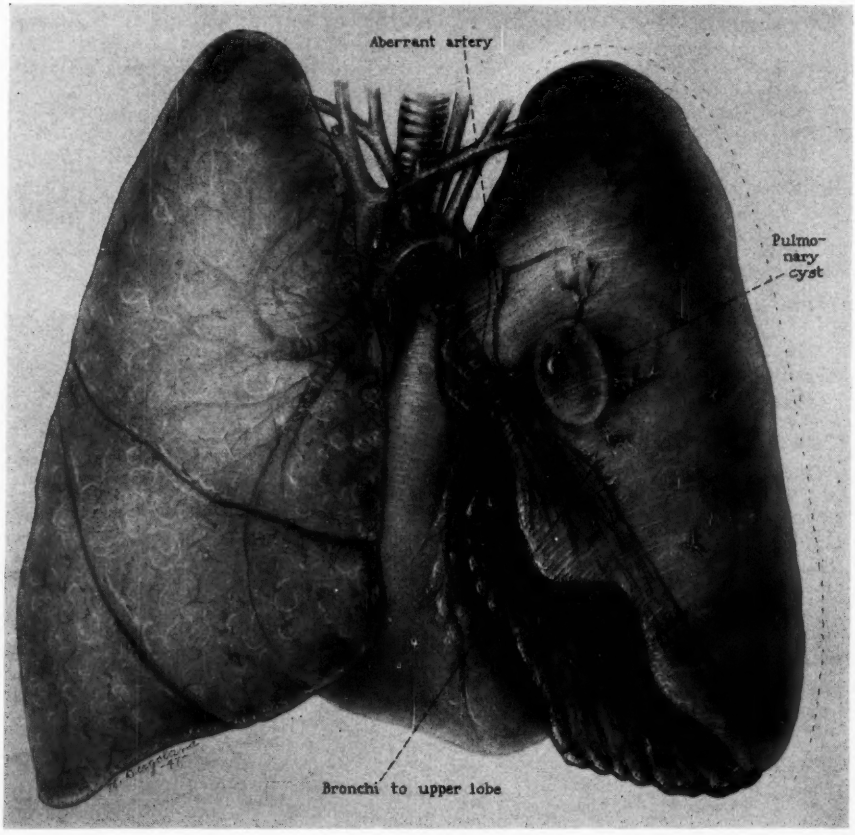

Fig. 6.—Illustrating atelectasis of left lower lobe and mediastinal shift secondary to emphysematous left upper lobe. Note aberrant artery and bronchi to upper lobe.

A diagnosis was made of pulmonary cyst of the left upper lobe with bronchopleural fistula and compression atelectasis of both lobes. It was felt that this cyst was sustained by a "ball-valve mechanism" in the communicating bronchi, and thoracotomy was undertaken in an effort to resect the cyst.

On May 21, 1947, under oral-intratracheal ether-oxygen anesthesia through a posterolateral incision with resection of the fifth rib and section of the fourth and sixth at the tubercle, the left thoracic cavity was opened. A minimal pneumothorax under slight positive pressure was found. The left upper lobe (Fig. 6) filled 80 per cent of the left thorax

and obviously was exerting pressure on the atelectatic, dark, fibrotic lower lobe. Re-expansion of this lower lobe was impossible even with 20 cm. pressure exerted through the intratracheal tube. The upper lobe was pink and though spongy in consistency it still was tense and remained so even after the manipulation attendant on pneumonectomy. Increasing the intratracheal pressure increased slightly the tenseness of this upper lobe. The surface of the lower lobe had intercommunicating lobule markings strongly outlined with anthracotic pigment; however, these markings in the upper lobe were much more widely separated and were a bright pink in color. The heart and mediastinum appeared normal. In the hilus of the lung there were several soft lymph nodes measuring up to 1 cm. in diameter; the pulmonary artery and veins were normal in size and position. An aberrant artery 3 mm. in diameter originated from the aorta near the origin of the second left intercostal artery and was directed into the mediastinal aspect of the upper lobe. Total pneumonectomy was performed by individual suture-ligation silk technique, and the thorax was closed without drainage. Convalescence was uneventful, and the patient was discharged on the twelfth postoperative day; a chest roentgenogram taken twelve weeks later (Fig. 7) revealed a satisfactory position of the heart and mediastinum. The patient returned to school where he was found to have a much greater exercise tolerance; the "cardiac consciousness" was gone.

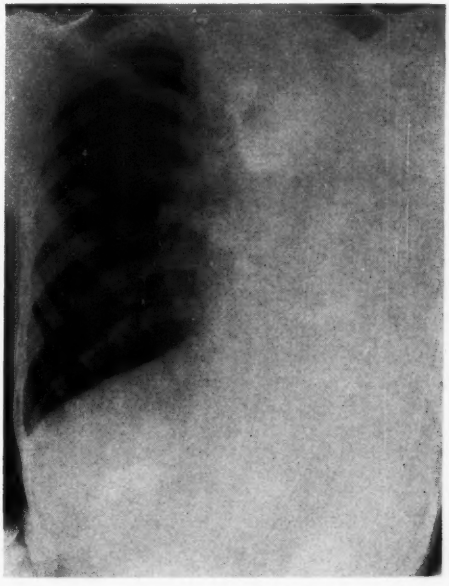

Fig. 7.—Anteroposterior roentgenogram taken twelve weeks postoperatively

Pathology.—The lung measured 6 by 12 by 17 cm.; the pink, emphysematous upper lobe was 5 by 12 by 13 cm. and had the form of a normal but greatly distended lobe. The pulmonary veins were normal in distribution. The pulmonary artery contributed several fibrinous branches to the upper lobe and then ramified throughout the lower lobe. The aberrant artery from the aorta branched soon after entering the upper lobe. The left stem bronchus entered only the lower lobe where it divided into two main trunks (Fig. 3). Just beyond this bifurcation the cephalad of these bronchi gave off three bronchi measuring about 2 mm. in diameter; these were identified as Bronchi 1 and 3 on the bronchodiagram. They extended across the interlobar fissure for about 2 cm. into the substance of the upper lobe where they terminated, patent, in the emphysematous tissue of the lobe. These were the only communications of the tracheobronchial tree with the upper lobe.

Cut section of the upper lobe revealed thin-walled emphysematous blebs throughout, 5 to 10 mm. in diameter. On microscopic examination (Fig. 8) the presence of such tissue was confirmed, and a diminished number of terminal bronchi were seen. Except for the previously described bronchi entering the lobe from the lower lobe there were seen but four or five bronchi, 2 to 3 mm. in diameter, and these radiated into the periphery of the lobe from a hilar cyst 1 by 1 by 2 cm. (Figs. 1 and 6). The cyst wall was firm, 3 mm. thick, and contained gray, inspissated mucoid material; microscopic examination of its wall (Fig. 9) revealed the structure of a large bronchus.

The lower lobe was atelectatic (Fig. 10), and cut section revealed an inordinate number of closely crowded bronchi. Detailed examination of the distribution of these bronchi confirmed the presence and arrangement of bronchi as represented on the composite bronchodiagram (Fig. 5).

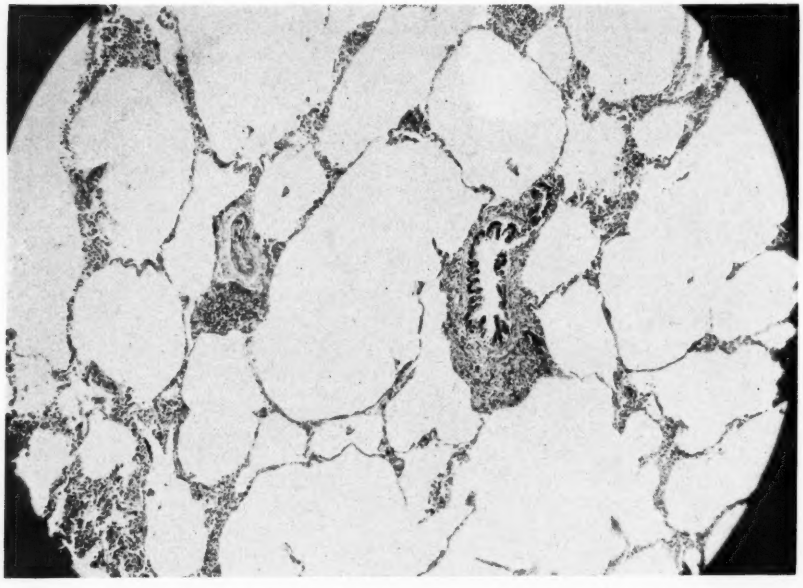

Fig. 8.—Illustrating bronchus and generalized emphysema of upper lobe (×79).

COMMENT

It has been assumed that the mesenchymatous anlage of a normal left lung gave rise to the two lobes of this specimen. The presence in the lower lobe of all the segmental bronchi normally found in an entire left lung has been interpreted as an ectopia bronchi of the left upper lobe. The posterosuperior segmental bronchi aerated but could not return air from the emphysematous, functionless upper lobe. Compression atelectasis of the lower lobe and mediastinal shift ensued. In the absence of a major bronchial communication from the upper lobe to the main bronchus it is impossible to explain the presence in that lobe of bronchi and of the bronchial cyst by the theory of lung formation propounded by Ekehorn[2] and Flint.[3] They described the development of branch bronchi as proceeding peripherally from the stem bronchus by a process of

Fig. 9.

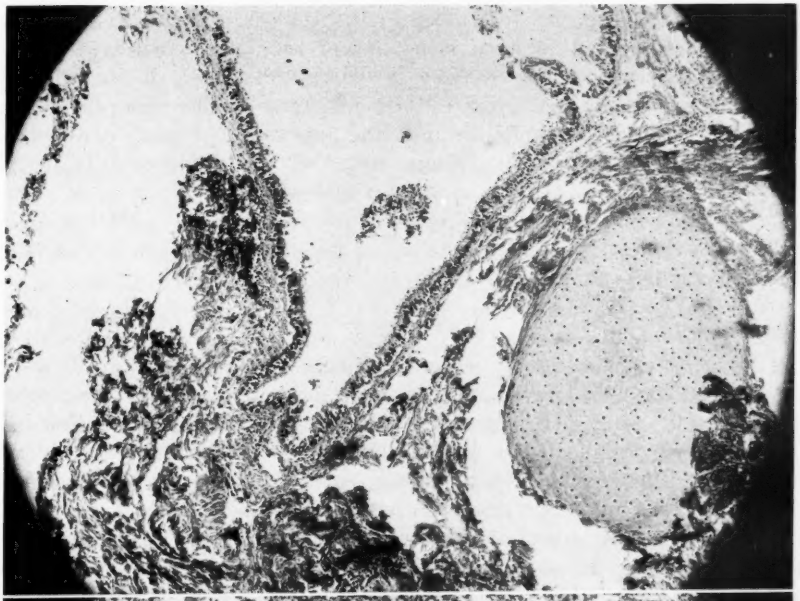

Fig. 10.

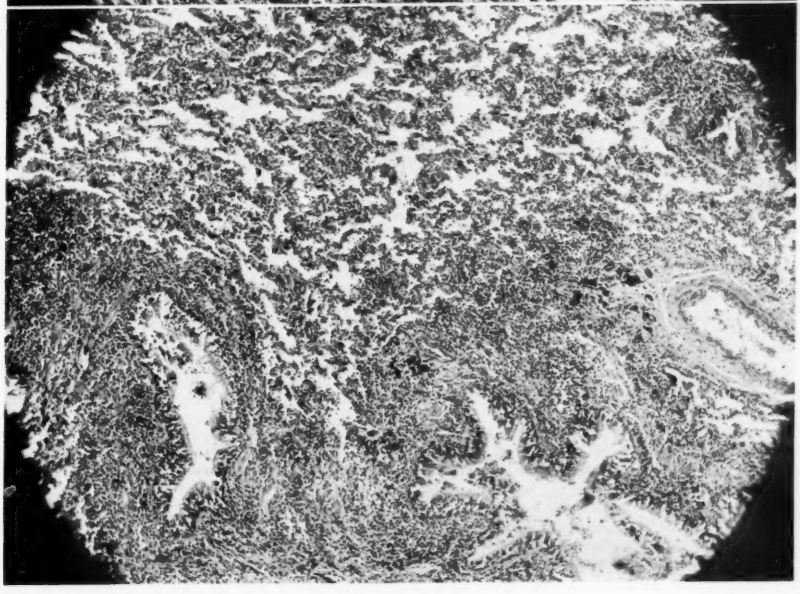

Fig. 9.—Illustrating bronchial epithelium and cartilaginous plate in wall of upper lobe cyst (×79).

Fig. 10.—Illustrating closely crowded bronchi and severe degree of lower lobe atelectasis (×79).

arborization. Heiss'[4] theory considers the stem bronchi as originating in the periphery of the lung and by a process of continuous infolding as giving rise to the connecting and terminal structures of the lung parenchyma. Such peripheral development has occurred in the case described; it but remained for the large bronchi arising centrally to join these smaller elements to complete a normal bronchial architecture of the upper lobe. Indeed, it would appear that here the mesenchymatous mass that Arey[1] denoted as contributing the nonepithelial elements to a lung had by a process of metaplasia given rise to bronchial epithelium. A more likely explanation is that the epithelium of the cyst and small bronchi arose from the bud of the left upper lobe bronchus, which bud later was incorporated into the lower lobe.

CONCLUSION

A case of ectopia bronchi of the left lung has been presented. Reference has been made to the prevailing theories of the development of the tracheobronchial tree and of the lungs, and the adequacy of these hypotheses has been questioned, particularly when reviewed in an effort to explain the anomaly described. If the knowledge of pulmonary embryology and the ability to diagnose lower respiratory tract anomalies is to keep abreast of recent advances in anatomy and surgery, detailed consideration must be given to a collection of many case reports such as that herein presented.

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LUNG HERNIA

A Case Report and Review of Literature

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THE occurrence of a case of lung hernia on the surgical service of this hospital has stimulated me to review the available literature. Lung hernia is a relatively rare condition. Less than 200 cases of lung hernia have been reported in the literature. A number of the earlier cases cannot be considered as true hernias of the lung but were merely traumatic eviscerations or prolapse.

A true lung hernia is defined as a protrusion of the lung and its pleural membranes through an abnormal opening in the thoracic enclosure.

The first case of lung hernia was reported by Roland in 1499.[9] However, this probably was not a true hernia of the lung. Montgomery and Lutz[9] credited Plater with the first report of a case of true lung hernia in 1641. Since that time there have been scattered reports of individual cases in the literature. Up to 1800 only fifteen cases had been reported, nine of which were true lung hernias. Morel-Lavalleé[9] in 1849 compiled a detailed study of the cases, described the symptoms, and classified pulmonary hernias.

In 1925, Montgomery and Lutz in an extensive review of the literature found 165 cases. Goodman[3] in 1933 compiled 171 cases from the literature. Maurer and Blades[8] made the most recent study of lung hernia and reported seven additional cases—the largest individual report in the recent literature.

CASE REPORT

Mr. L. C. B., aged 46 years, a steamfitter, entered this hospital on March 28, 1946, with a history of having been in an automobile accident at 6 A.M. March 27, 1946. On admission the patient was found to have multiple lacerations and abrasions about the face, head, and left side of the chest; marked subcutaneous emphysema of the chest wall and supraclavicular areas; and multiple fractures of the left sixth, seventh, eighth, and ninth ribs posteriorly. There was considerable displacement of the fractured segments of the sixth and seventh ribs. A left hemothorax and hemopericardium ensued but under conservative therapy spontaneous recovery occurred except for slight pain and tenderness in the region of the fractured ribs.

On Jan. 16, 1947, the patient was readmitted to this hospital complaining of pain in the left posterior portion of the chest. The patient stated that following previous hospitalization he was unable to work because of pain. The pains were described as beginning beneath the left scapula and radiating laterally to the left shoulder. The pains were burning in character, constant, and aggravated by coughing. The patient also complained of dyspnea and slight hemoptysis on several occasions.

Physical examination on admission revealed a well-developed and well-nourished white man, ambulatory and not acutely ill. Temperature was 98.6° F., pulse 100, and respiration 20. The right pupil was contracted and the left pupil dilated; pupillary reflexes were absent. There was marked dental caries. The chest was barrel-shaped; a depression was noted beneath the inferior angle of the left scapula over the sixth and seventh ribs posteriorly. The depression was partially covered by the scapula. There was a palpable defect in the chest

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cage in this area measuring approximately 3 inches in diameter. On coughing with the scapula abducted and rotated there was an expansile bulge measuring about 4 by 5 inches through the defect in the chest cage. Coarse râles were noted in the right upper lung fields. There was some pain in the left shoulder on motion with audible crepitations but no limitation of passive motion. The remainder of the physical examination was entirely normal except for a healed scar on the glans penis. Blood pressure was 130/85.

Laboratory findings were red blood count, 5,460,000; hemoglobin, 15.23 Gm.; white blood count, 10,450. Differential white count was within normal limits, Wassermann and Kahn tests 4 plus, urine negative.

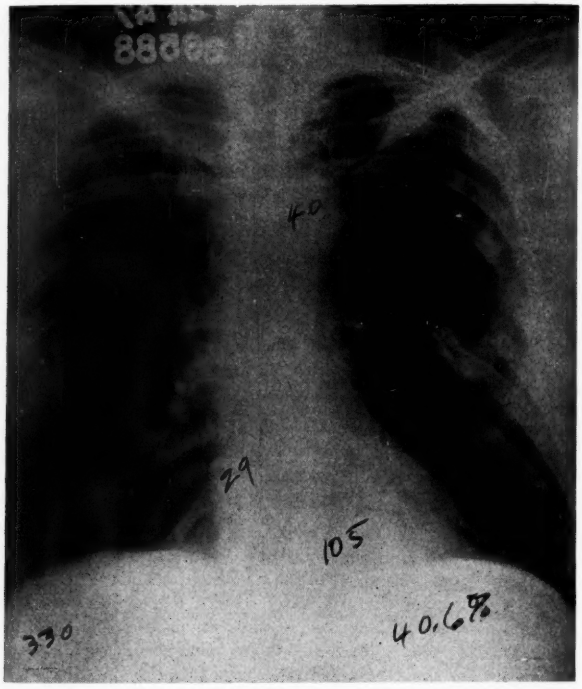

Fig. 1.—Chest x-ray picture taken Jan. 24, 1947, showing multiple fractures of the sixth, seventh, eighth, and ninth ribs on the left. Displacement of the fragments of the sixth and seventh ribs is noted, producing a defect through which herniation of the lung occurred.

X-ray findings revealed chronic hypertrophic arthritis in the left shoulder and lower thoracic spine. There were deforming fractures involving the posterior aspects of the sixth, seventh, and eighth ribs (Fig.1). The sixth rib was fractured at the posterior axillary and at the paravertebral lines and the outer portion was displaced upward. The seventh rib was similarly fractured with the outer portion displaced downward. The eighth rib was fractured in the mid-posterior portion. Marked callus formation was noted at the fracture sites. The lung fields were clear and the cardiac shadow was within normal limits.

The electrocardiogram was interpreted as normal.

Clinical diagnosis was hernia of the lung, traumatic, acquired, left; chronic bronchitis; arthritis, chronic hypertrophic, left shoulder and lower thoracic vertebrae; syphilis, tertiary, latent.

Repair of the lung hernia was recommended and performed on Feb. 3, 1947.* Under

*Operation performed by Wayne C. Bartlett, M.D.

intratracheal anesthesia an incision was made over the seventh rib extending from the transverse process posteriorly to the posterior axillary line. The incision was extended down through the muscle and fascia until the pleura was reached. In portions the pleura was found directly beneath the skin. The hernial sac consisted of diverticular outpouchings of the pleura extending out over the posterior surface of the ribs. The sac was dissected free. The sixth rib was found to be fractured in two places and the distal end of the middle fragment was embedded in the pulmonary tissue underlying the fifth rib. This was freed and brought up into the operative field. The seventh rib was similarly fractured with the fragment displaced down under the eighth rib. This was likewise freed and brought up into the operative field. The eighth rib had been fractured but had healed with only a moderate amount

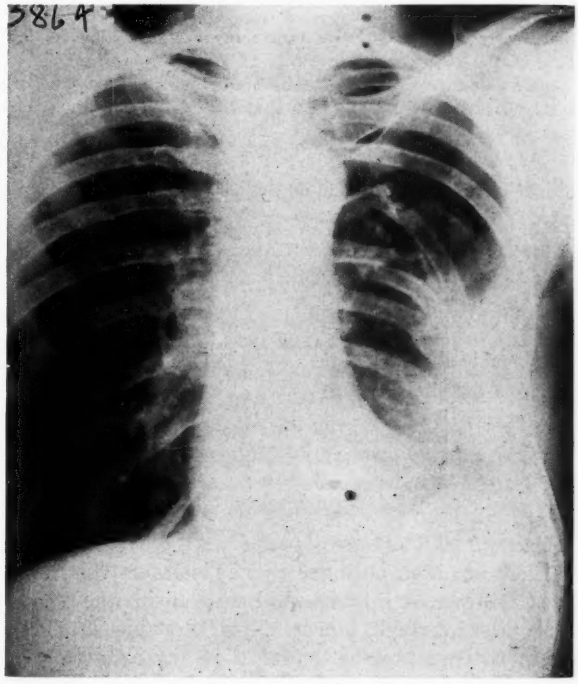

Fig. 2.—Postoperative chest x-ray view taken May 20, 1947. The defect in the thoracic cage has been corrected. Periosteal new bone formation is noted. There is mal-alignment of the fractures resulting from insufficient immobilization. Narrowing of the interspaces and thickening of the pleura in the left midportion and base are observed.

of deformity. Adhesions of the lung to the hernial sac were found and freed and the lung allowed to retract into the pleural cavity. The hernial sac was then removed and the edges of the pleura were approximated with No. 000 chromic catgut sutures. The ends of the seventh rib were freshened. Holes were bored into each end and a fine stainless steel wire was used to approximate them. The sixth rib was similarly treated. This brought the ribs fairly well into a normal alignment. The overlying muscles and fascia were then closed with interrupted chromic catgut sutures. The skin was closed with interrupted black silk sutures. The patient received 500 c.c. of citrated blood and 1000 c.c. of glucose in saline during the operative procedure.

Postoperative course.—Shortly after the patient's return to his room respiratory embarrassment was noted, necessitating water-trap suction to reduce excessive pneumothoax;

suction was discontinued after two hours. General supportive therapy was instituted and penicillin was administered intramuscularly every three hours until Feb. 22, 1947. A moderately severe persistent cough associated with moderate mucopurulent sputum and left chest pains were noted postoperatively. X-ray examination of the chest on Feb. 4 showed some fluid in the left chest. This increased in amount, necessitating the aspiration of 870 c.c. of serosanguineous fluid on Feb. 10, 350 c.c. on Feb. 13, and 50 c.c. on Feb. 26; 100,000 units of penicillin were instilled into the pleural cavity after each thoracentesis. The sutures were removed on Feb. 10 at which time the wound edges appeared slightly reddened and indurated. The patient was allowed up in a wheel chair on the same date and soon thereafter was ambulatory. He developed a laryngitis on Feb. 20 which was treated with penicillin inhalations and responded slowly. The wound was well healed and when last seen in June, 1947, there was no evidence of recurrence of the lung hernia. The patient has vague chest and shoulder pains which seem to be greatly exaggerated. Re-check x-ray views on May 20, 1947, reveal some residual thickening of the pleura with narrowing of the interspaces on the left; at the site of the hernia repair there is some displacement of the rib fragments and evidence of periosteal new bone formation (Fig. 2).

DISCUSSION

The classification of lung hernias as devised by Morel-Lavalleé in 1849[9] and accepted by most authors is as follows:

I. According to location:
   - A. Diaphragmatic
   - B. Thoracic
   - C. Cervical

II. According to etiology:
   - A. Congenital
   - B. Acquired
     1. Traumatic
     2. Consecutive
     3. Spontaneous
     4. Pathologic

Korol,[6] Hartung,[5] and others question the occurrence of diaphragmatic lung hernia. There has been only one case reported in the literature. Beale[1] in 1882 reported the case of a 22-year-old white man who was pinned to the ground by a cart wheel striking him in the right abdomen. There was no external wound but autopsy revealed a defect in the right diaphragm through which the lung had apparently herniated with strangulation and abscess formation. Korol[6] considered this case as one of prolapse rather than herniation. Because of the pressure relationships, herniation of the lung through the diaphragm appears unlikely.

Korol[6] included mediastinal hernia in the category of lung hernia. A mediastinal lung hernia is a herniation of the lung through the mediastinal septum into the opposite hemithorax. Korol stated that it is the most frequent form of lung hernia, although few cases are noted in the literature to confirm this. It is said to occur frequently following modern collapse therapy (Korol, Hartung). The herniation occurs either anteriorly in the space between the sternum and the heart at the site of the atrophied thymus gland, or posteriorly between the esophagus and aorta below the level of the trachea and arch of the azygos vein. Mediastinal hernias are brought on by emphysema of one lung associated with contraction, atelectasis, or absence of the opposite lung. It

may follow the abandonment of a long-continued pneumothorax. The pleural hernia of a tension pneumothorax is not to be confused with a mediastinal lung hernia.

Cervical lung hernias occur through the superior aperture of the thorax between the sternocleidomastoid and scalenus anticus muscles and are due to a tear or a deficiency of Sibson's fascia which normally limits the cervical excursion of the apex of the lung. Up to 1938 there had been only twenty cases reported in the literature.[6] They are usually said to be congenital.

The most common form of lung hernia is the thoracic or intercostal. Out of seventy-eight case reports reviewed by Montgomery and Lutz[9, 10] there were sixteen cervical hernias and sixty-two thoracic hernias. Thoracic hernias usually occur anteriorly near the sternum and posteriorly near the vertebrae. This is said to be due in each case to a locus minoris resistentiae in these areas, anatomically due to the fact that the external intercostal muscles extend forward only as far as the costochondral junctions and the internal intercostal muscles extend posteriorly only to the angle of the rib. Thoracic hernias are said to be more frequent anteriorly due to the fact that the pectoralis muscles cannot give as good support as the trapezius, latissmus dorsi, and rhomboideus muscles.

Montgomery and Lutz, in their review of 165 cases found in the literature, reported an incidence of 29 (18 per cent) congenital or early acquired hernias, 83 (50 per cent) traumatic, and 53 (32 per cent) spontaneous hernias. There has been some difficulty in determining whether a hernia is congenital or not. This is especially true when the hernia appears many months after birth. Hochsinger[11] stated that all hernias occurring in the first few weeks of life must be considered as congenital. Congenital lung hernias are due to several factors: (1) Weakness of the anatomic structures about the apex of the lung; (2) arrested growth of structures, that is, chest wall, sternal clefts; (3) intrauterine abnormalities such as pressure of an elbow against the fetal chest, amniotic bands, and pressure from tumors.

The great majority of acquired hernias are traumatic. A traumatic lung hernia may develop at any time after injury. Those first appearing late after injury have been classified by Morel-Lavalleé as consecutive hernias. Most writers agree that this subdivision is superfluous and merely group all such cases as traumatic hernias. Traumatic hernias may develop following operative wound, penetrating or bullet wound, or crushing injuries. Lung hernias are only infrequently noted following chest surgery. Careful subperiosteal resection of the ribs in thoracoplasty and other chest surgery probably accounts for the rarity of this complication.

Spontaneous lung hernias are the result of two factors, namely, a locus minoris resistentiae (that is, an area of local decreased resistance in the boundaries of the chest cavity) and an abnormal increase in the air pressure in the alveoli of the lungs. The abnormal increase in the intrathoracic pressure may be due to various factors. Paroxysmal coughing, which may be due to a variety of conditions such as chronic bronchitis, bronchiectasis, whooping cough, pulmonary abscess, and foreign bodies of the lung, is an important etiologic factor.

Occupations, such as glass blowing and playing of wind instruments, have been found etiologically responsible for a number of cases. Violent muscular exertion, such as heavy weight lifting or childbirth, is a third factor in the occurrence of lung hernias.

Pathologic lung hernias result from a variety of diseases of the chest wall which produce a locus minoris resistentiae. Abscess of the chest wall and breast, perforating lung abscess or empyema necessitatis, and malignancies have all been responsible for the occurrence of pathologic lung hernias.

According to Montgomery and Lutz, lung hernias are three times more frequent in males. Other authors report an even greater incidence in males.

The symptoms of lung hernias are varied. Goodman[3] listed three cardinal symptoms as follows: bulge, cough, and pain at the site of the bulge. The onset is usually insidious accompanied by pain and cough. The cough is chronic, spasmodic, and may or may not be productive depending on the underlying pathology. Early, the traumatic hernias are usually obscured by the more severe signs and symptoms of injury. The hernia may rarely become incarcerated or strangulated producing inflammatory changes.

On physical examination there is noted a bulging mass which increases on forced inspiration. A defect in the chest wall is usually readily demonstrable. When the hernia is protuded, the soft spongy crepitant consistency of the lung may be noted. A scar may or may not be present depending on the etiology.

X-ray and fluoroscopy may be of assistance in demonstrating the defect of the chest wall and an area of increased translucency may be noted outside the normal pulmonary boundaries. In mediastinal hernias there may be an asymmetry of the chest and on x-ray examination a large hyper-aerated lung is seen to extend into the opposite side in one or two bulgings having definite convex outer borders. The upper bulge is anteriorly between the first and fourth ribs and the lower bulge is posteriorly between the fifth rib and diaphragm. The trachea and esophagus are displaced to the contracted side.

The diagnosis of lung hernia is not difficult if the condition is kept in mind. The physical findings are readily apparent. The differential diagnosis should be made from subcutaneous emphysema, perforating empyema, aneurysm of the intercostal vessels or aorta, fibroma, lipoma, and hemangioma. Mediastinal lung hernias must be differentiated from the pleural hernia of a tension pneumothorax in which there is no lung herniation present.

Complications are rare. Strangulation and gangrene of the lung may occur. Hemoptysis is not infrequent. The prognosis of lung hernia is good; it is rarely fatal, but rarely heals spontaneously.

The treatment of lung hernias is either medical or surgical. Medical treatment is in the main palliative consisting of rest, change of occupation, and local compression with pads and elastic devices. With palliative treatment the defect in the chest wall may become increasingly rigid, but the hernia, although smaller, will still persist; an increase in intrathoracic pressure will cause a return and increase in the size of the protusion. Surgery is contraindicated in a number of cases due to the underlying pulmonary pathology and general condition of the patient.

Surgery offers the only hope for a permanent cure. Various operative procedures have been devised. Vogler[8] in 1898 performed a plastic operation using periosteum or bone flap from the sternum. Vulpius[8] in 1898 performed a plastic operation in which he crossed strips of ribs which were sutured in place with silver wires; this hernia recurred in one year. Tuffier[8] in 1891 reported a cure by freeing and ligature of the hernial sac. Graham[4] in 1922 treated a spontaneous cervical hernia associated with an adenoma of the thyroid by excision of the adenomatous thyroid and packing of the wound with iodoform gauze. The packing was removed on the fifth postoperative day and the wound healed without recurrence of the hernia. Montgomery and Lutz[9] in 1925 reported a case of traumatic intercostal hernia cured by an osteoplastic procedure.

Goodman[3] in 1933 treated a traumatic consecutive intercostal hernia in a 42-year-old seaman by periosteal plastic closure. The redundant pleura was resected and closed with continuous catgut suture following which the periosteum of the rib above the defect was reflected downward, the periosteum of the rib below the defect was reflected upward, and both were sutured over the defect with chromic catgut.

Winkel[14] in 1935 reported a cure in a traumatic intercostal hernia by a repair consisting of weaving of fascia lata strips across the defect.

Repair of the defect with tantalum plate has been attempted by Paulson[8] but has been found unsatisfactory because of difficulty of immobilization of the chest wall.

These have in the main been individual case reports. Maurer and Blades[8] have undoubtedly had the largest individual experience with the surgical treatment of lung hernias in recent times. In 1946 they reported seven cases of traumatic intercostal lung hernias, four patients of whom submitted to surgery consisting of closure by periosteal flaps. In addition, they reported four cases of herniation of the liver and peritoneum through the lower thoracic cage which were repaired in a similar manner. Results have been excellent. Maurer and Blades believe that any defect of the chest wall regardless of size can usually be repaired by plastic procedures utilizing only structures which are a part of the chest wall, namely the rib, periosteum, and muscle. The most important feature is closure of the defect with sturdy bone or periosteum which will produce bone. The freeing of adhesions and of the lung about the margins of the defect with the creation of a local pneumothorax is important in the production of a cushion between the lung and the freshly repaired chest wall. This serves to reduce to a minimum the possibility of the lung being forced into the chest wall by coughing and straining during the immediate postoperative course.

SUMMARY

A review of the available literature concerning lung hernias has been presented.

A true lung hernia is a protusion of the lung and its pleural membranes through an abnormal opening in the thoracic enclosure. Its classification, etiology, symptoms, and treatment have been reviewed. The diagnosis is not difficult provided the condition is kept in mind.

A case report of an acquired traumatic lung hernia discovered ten months after injury has been presented. Repair by osteoplastic procedure as outlined by Montgomery, Goodman, Maurer, and Blades, and others was not feasible because of the multiple rib fractures. An apparent cure was obtained by the excision of the hernial sac, realignment and fixation of the fractured ribs, and reapproximation of the muscles and fascia.

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IMMEDIATE CARDIOVASCULAR AND RESPIRATORY ADJUSTMENTS TO PNEUMONECTOMY

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THE details of the physiologic adjustments of the cardiovascular and respiratory systems to the removal of one lung are of increasing importance in view of the frequency with which pneumonectomy is being performed today.

The effect of abrupt occlusion of one lung root was studied as early as 1876 by Lichtheim.[1] Later Gerhardt,[2] Plumier,[3] and Welch[4] investigated the effects on systemic blood pressure of occlusion of one main branch of the pulmonary artery. It was found that in dogs arterial blood pressure was not appreciably altered by these procedures. In 1932, Gibbons, Hopkinson, and Churchill[5] found that circulatory failure followed the abrupt occlusion of 60 per cent or more of the pulmonary artery and felt that the failure was due to a reduced volume of circulating blood. In 1936, Fineberg and Wiggers[6] studied the effect of the partial occlusion of the pulmonary artery in dogs on the pressure pulses of the right ventricle. Compensation of the right ventricle to increased resistance was judged to be complete so long as the left ventricular output remained unaffected, as indicated by unaltered aortic pressure curves. They found that the right ventricle could compensate completely when an average of 58 per cent of the artery was occluded for 3 to 5 minutes. Prolonged compression at this degree of partial occlusion or increased occlusion of the artery resulted in circulatory failure which Fineberg and Wiggers felt was due to fatigue of the musculature of the right ventricle. The effect of pneumonectomy on the blood flow through the lungs in dogs was studied by Andrus,[7] who found that within a few hours after operation the blood flow through the one remaining lung exceeded the total flow through both lungs preoperatively. This increased cardiac output was found to persist about ten days.

The present acute experiments on dogs with closed chests are concerned with the immediate cardiovascular and respiratory effects of sudden ligation of an entire lung root. In most instances the right lung has been selected for experiment.

PROCEDURE

Mongrel dogs were prepared twenty-four to forty-eight hours before the acute experiment. Under nembutal anesthesia (35 mg. per kg.) the chest was opened aseptically. Artificial respiration was used. The pulmonary ligament was cut and a linen ligature placed around the lung root. The ligature was tied in a loose slip knot, and the long end was brought out through the lateral incision in the chest wall. The lung was fully inflated with positive pressure, and the chest wall tightly closed.

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The acute experiment was performed under sodium pentothal-sodium barbital anesthesia (sodium pentothal 20 mg. per kg., sodium barbital 150 mg. kg.). A rubber catheter was passed through the external jugular vein into the right ventricle under fluoroscopic guidance. At the time the dog was under the fluoroscope the state of the heart and lungs was noted. In no animal was there an accumulation of pleural fluid. Residual pneumothorax was minimal.

Right intraventricular pressure was measured through the intracardiac catheter by a water manometer; mean systemic blood pressure was measured from a cannula in a femoral artery. Cardiac output was estimated by the direct Fick principle, mixed venous blood being withdrawn from the catheter in the right ventricle and arterial blood being taken from the uncannulated femoral artery by needle puncture. Blood oxygen content was determined in duplicate by the method of Van Slyke and Neill. Oxygen consumption was measured continuously with a modified Roth-Benedict spirometer. An endotracheal catheter attached directly to the spirometer eliminated the necessity for tracheotomy. Leakage of air around the catheter was prevented by inflating a thin rubber cuff fitted to the outside of the catheter. This method made it possible to record any changes in tidal air and rate of respiration as well as oxygen consumption.

Records were made until the dog was in a steady state, usually one to two hours after the anesthesia had been given. Simultaneous arterial and right ventricular blood samples were then collected for estimation of the cardiac output. The ligature about the lung root was tightened by pulling on the end protruding through the chest wall. Occlusion was complete in 4 to 8 seconds. During this period continuous recordings were made of oxygen consumption, tidal volume, right intraventricular pressure, systemic blood pressure, and Lead II of the electrocardiogram. Within 5 minutes of ligation samples of blood were again drawn for the second cardiac output determination. The recordings were continued one to two hours or until the animal died.

A total of twenty dogs were studied. Autopsies were done in all cases. The condition of the lungs, the effectiveness of the ligature about the lung root, and the state of the heart were observed. In all instances the ligature effectively occluded the bronchus and accompanying blood vessels.

RESULTS

The sudden ligation of one lung root caused an immediate rise in right intraventricular pressure of from 20 to 129 mm. of water (average 57 mm.) lasting 3 to 60 minutes (average 25 minutes). There was a transient slowing of the heart for 4 to 5 beats but no other change in the electrocardiogram. Systemic blood pressure showed a transient fall of 10 to 15 mm. Hg during the time the ligature around the lung root was being tightened but rose again to the preligation level and remained steady (Fig. 1).

In eight dogs the changes in cardiac output after ligation of the lung root were no more than ± 200 c.c. per minute (from ± 5 to 17 per cent of the control value), and these were considered insignificant variations. (Significance was based upon both the limits of accuracy of the method for determination of

oxygen content of the blood samples and changes in output which might reasonably be expected to occur over a ten-minute period under barbiturate anesthesia.[9, 10]) In three dogs there were increases in cardiac output of 400 c.c., 800 c.c., and 1,900 c.c. per minute (+ 25 per cent, 32 per cent, and 63 per cent). In six instances the output decreased 300 c.c. to 700 c.c. per minute (18 to 45 per cent), and in two the outputs fell 1,600 c.c. and 1,900 c.c. per minute, decreases of 53 per cent and 68 per cent.

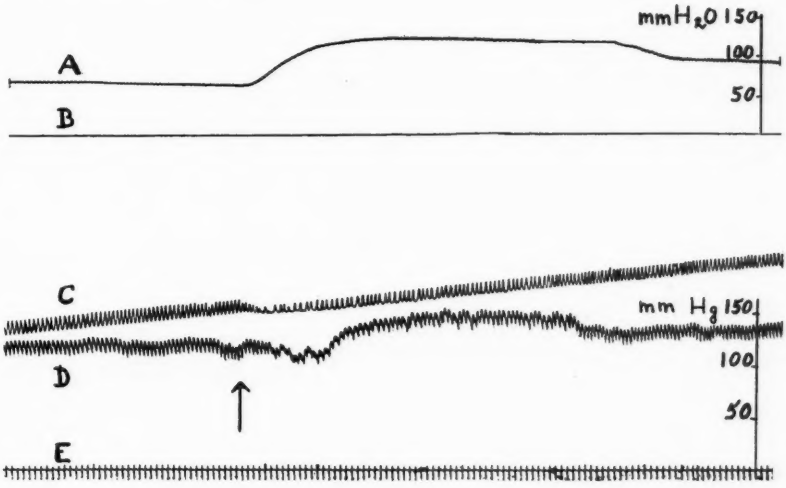

Fig. 1.—Effects of ligation of right lung root (Dog 16) at arrow. *A*, Right intraventricular pressure; *B*, base line at level of right atrium; *C*, respiration and oxygen use; *D*, mean arterial blood pressure (femoral artery); *E*, base line for blood pressure, 4 second intervals.

The oxygen consumption before and after ligation remained essentially the same. In twelve dogs the minute volume of respiration did not change significantly; in four of these neither the tidal air nor the rate of respiration changed, and in eight an increase in rate of respiration was proportional to a decrease in tidal air. (Significance was based on the fact that, with the spirometer used, tidal air could be estimated within 10 c.c. A change in minute volume of less than ten times the rate of respiration was considered insignificant.) The minute volume decreased 300 to 800 c.c. (19 to 25 per cent) in four dogs; in three of these this was due to a decrease in tidal air without a completely compensatory increase in rate, and in one exceptional case in which respiration became suddenly slower and deeper the decrease in rate (41 per cent) was greater than the increase in tidal air (37 per cent). The minute volume increased 1,000 to 1,100 c.c. (30 to 44 per cent) after ligation in four instances due to a very large increase in rate of respiration (25 to 59 per cent) out of proportion to any change in tidal air.

In the twelve dogs with no significant change in over-all minute volume there was no significant change in the efficiency of respiration as measured by the ventilation equivalent. (Ventilation equivalent = liters of air necessary to be

ventilated for the absorption of 100 c.c. of oxygen.[8]) With an increase in minute volume, the efficiency decreased (ventilation equivalent increased); with a decrease in minute volume efficiency of oxygen absorption increased (ventilation equivalent decreased).

TABLE I. CARDIOVASCULAR FUNCTION BEFORE (A) AND AFTER (B) PNEUMONECTOMY

NO.	LUNG	CARDIAC OUTPUT (LITERS PER MIN.) A	B	PER CENT CHANGE	STROKE VOL. (C.C.) A	B	PULSE A	B	RT. VENT. PRESS. MM. OF H_2O SYSTOLIC	TIME TO RETURN (MIN.)	ART. B.P. CHANGE	AUTOPSY ABNORMAL FINDINGS
1	RPA	1.9	2.0	+ 5								
2	LL	1.2	1.0	−17								
3	RL	1.9	1.8	− 5								RLL abscess
4		2.5	3.3	+32								
5	RL	0.7	0.4	−42	5.4	2.7	140	148				
6	RL	1.1	0.6	−45	6.5	3.2	168	190				
7	RL								28	8		
8	RL	2.1	1.9	− 9	17.5	16.5	120	114	81	60		LUL, LML atelectatic
9	LL	3.0	4.9	+63	18.7	36.2	160	135	36	15		LLL atelectatic
10	LL	2.2	2.1	− 5	14.5	14.0	160	150	None			
11	RL	0.5	0.3	−40	3.8	2.1	130	140	70	30	No change	RLL, atelectatic
12	RL	1.2	1.2	0	6.3	7.6	190	170	80	30		
13	RL	2.8	0.9	−68	16.5	5.3	170	170	105	30	Fell, Died	Infarct RLL, RML, worms right heart
14	RL	1.8	1.3	−27	9.5	8.1	190	160	20	15		Infarct RLL
15	RL	2.1	1.9	− 9	10.0	7.6	210	250	55	30	Sl. fall	
16	RL	3.8	3.1	−18	23.4	17.2	162	180	117	25	No change	Infarct RLL
17	RL	3.0	1.4	−53	20.0	10.0	150	140	59	60	Fell	
18	RL	1.5	1.9	+26	10.0	12.6	150	150	40	17	No change	
19	RL	2.2	1.7	−23	15.7	14.2	140	120	40	7	Sl. rise	Catheter Rt. atrium
20	RL	1.2	1.3	+ 8	7.6	8.7	170	150	20	3	No change	

At the end of the acute experiment eighteen of the twenty dogs had apparently compensated for the functional removal of one lung. The blood pressure had been maintained and there was no respiratory difficulty. Two failed to compensate. One of these showed a decrease in cardiac output from 2.8 to 0.9 liters per minute, the stroke volume falling from 16.5 to 5.3 c.c. Systemic blood pressure fell immediately from 72 mm. Hg to 54 mm. Hg, rose to 86 mm. and then fell gradually until death occurred at the end of one hour. At autopsy the heart was found to be dilated and the right heart and pulmonary artery filled with the round worms *Dirofilaria immitis*. On section the blood vessels in the lung were also found to be full of microfilariae of *D. immitis*. The cardiac output of the second dog fell from 3.0 to 1.4 liters per minute, the systemic blood pressure decreased gradually from 130 to 64 mm. Hg, and Traube-Hering waves appeared. After two hours the animal appeared unlikely to recover and was sacrificed. At autopsy the heart and lungs were grossly normal.

DISCUSSION

The conditions under which the experiment was carried out were admittedly not entirely normal. Barbiturate anesthesia is known to decrease the cardiac

TABLE II. PULMONARY FUNCTION BEFORE AND AFTER PNEUMONECTOMY

NO.	LUNG	O_2 USE C.C. PER MIN.		MIN. VOL. LITERS PER MIN.		TIDAL AIR (C.C.)		RESP. RATE PER MIN.		VENT. EQUIV. L. PER 100 C.C.		AUTOPSY, ABNORMAL FINDINGS
		BEFORE	AFTER	BEFORE	AFTER	BEFORE	AFTER	BEFORE	AFTER	BEFORE	AFTER	
1	RPA	62	62	4.5	4.5	42	42	108	108	7.3	7.3	
2	LL	35	35	3.7	4.7	83	52	45	88	1.1	1.3	
3	RL	42	44	2.7	2.7	71	71	38	38	6.4	6.1	RLL Abscess
4		56	62	2.5	3.6	67	73	38	49	4.5	5.8	
5	RL	52	53	3.6	3.2	83	54	43	59	6.9	6.0	
6	RL	30	30	1.6	1.3	72	52	22	25	5.3	4.3	
7	RL	46	43	2.0	2.2	100	100	20	22	4.4	5.1	
8	RL	60	67	3.4	3.0	62	52	55	57	5.7	4.4	LUL, LML atelectatic
9	LL	74	74	3.7	3.0	83	114	44	26	4.9	4.0	LLL atelectatic
10	LL	53	53	2.8	3.8	104	98	27	39	5.3	7.2	
11	RL	25	25	3.6	4.7	108	93	33	50	1.4	1.9	RLL atelectatic
12	RL	52	59	2.7	3.8	93	83	29	46	5.2	6.5	
13	RL	64	57	3.4	3.0	89	72	38	42	5.3	5.3	Infarct RLL, RML, worms right heart
14	RL	49	46	1.9	1.3	62	39	30	32	3.8	2.7	Infarct RLL
15	RL	242	242	3.7	4.3	62	55	60	78	1.5	1.8	
16	RL	83	85	3.0	3.2	166	145	18	22	3.6	3.8	Infarct ½ RLL
17	RL	62	55	1.3	0.7	35	13	36	51	2.0	1.2	
18	RL	83	83	3.2	2.4	83	52	39	46	3.9	2.9	
19	RL	83	71	2.2	2.0	42	42	53	48	2.7	2.8	
20	RL	73	80	3.6	3.7	125	93	29	40	5.0	4.7	

output of an animal.[9, 10] According to Shore and associates[9] the decrease in cardiac output with anesthesia is neither constant nor predictable. Over 20 to 30 minute periods they cite decreases of from 10 to 30 per cent, and, on the other hand, over some 90 minute periods they have found decreases of only 6 to 7 per cent. In our studies the two estimations of cardiac output were made not more than 10 minutes apart, and at least 60 minutes after induction, hence, since the animal was in a steady state with regard to blood pressure and oxygen consumption when the ligature was tightened, it may be assumed that any change in cardiac output more than 10 per cent in the period immediately following could be attributed to the manipulation.

The experiments were done with the chest closed to enable a continuous recording of both oxygen consumption and the details of pulmonary ventilation. The dog was breathing against a definite resistance at all times, but this resistance was constant, and relative changes in the character of respiration were readily seen.

Since we found the right pulmonary artery to be slightly larger in diameter than the left, the right side was used after the first few experiments as the side which might be expected to give the greater changes.

Compensatory circulatory and respiratory mechanisms were sufficient in all but two dogs to maintain the systemic blood pressure and the oxygen consumption virtually unchanged. This speaks for the large reserves of the vascular bed and airways of one lung which can immediately take over the functions of two.

The outstanding change in the cardiovascular system, which occurred in all but one instance, was an increase in right intraventricular pressure. This rise was relatively slow in development, taking from 2 to 10 minutes to reach its peak, remaining on a plateau for 10 to 15 minutes, and gradually falling to the preligation level in 25 to 30 minutes. It was not correlated with changes in cardiac output, stroke volume, or systemic blood pressure. One might expect that the sudden removal of a large part of the pulmonary capillary bed would result in greatly increased resistance in the pulmonary circuit. The return of the ventricular pressure to its former level suggests that additional vascular channels were opened, or that existing vessels increased in size under the increased pressure. This opening of a reserve vascular bed has been postulated previously by Tigerstedt[11] and by Wiggers.[12]

The cardiac output following compression of the pulmonary artery has been of interest to many observers. In 1907 Tigerstedt,[11] measuring the minute output of the left heart with a stromuhr, found that it was only slightly decreased by ligation of the left pulmonary artery. Haggart and Walker,[13] in nine experiments on cats, also found only a slight decrease in cardiac output with compression of the left pulmonary artery. Fineberg and Wiggers[6] found decreases in left ventricular output with compressions of the pulmonary artery exceeding 41 to 72 per cent (average 58 per cent).

In the present studies there was no change in cardiac output in eight experiments. In three the output increased. In eight there was a significant

decrease in cardiac output following ligation of a lung root. With a decrease in cardiac output up to 45 per cent, compensatory mechanisms were adequate to prevent a change in systemic blood pressure. In the two cases in which the cardiac output fell 53 and 68 per cent the arterial blood pressure fell steadily over the remainder of the experimental period.

The changes in cardiac output, when they occurred, appeared to depend largely on changes in stroke volume. Although the heart rate was usually somewhat altered by ligation of the lung root (average change plus or minus 19 beats per minute), the direction of change was unpredictable and the magnitude not sufficient in itself to effect a significant increase or decrease in cardiac output if not associated with a similar change in stroke volume. Out of a total of fifteen dogs, eight showed changes in stroke and pulse opposite in direction; five resulted in significant changes in output in the direction of the change in stroke volume. Two dogs maintained a constant heart rate but variations in stroke volume were sufficient to bring about significant alterations in cardiac output. In three instances in which there was a significant decrease in cardiac output, changes in stroke volume and heart rate were parallel.

With ligation of one lung root a certain volume of blood is trapped in the lung. What part this plays in the reductions of cardiac output in these experiments is not known.

There was a transient drop in systemic blood pressure which occurred as the ligature was being pulled tight. Parin[14] has described a fall in systemic blood pressure which he believes due to a reflex mediated through the vagus nerve from pressoreceptors in the pulmonary artery. The pressures used by Parin experimentally were 40 mm. of Hg or above. In the present studies the pressures in the right ventricle, and therefore in the pulmonary artery, did not approach 40 mm. of Hg. Furthermore, the transient fall in blood pressure did not coincide with the rise in right intraventricular pressure but preceded it. The transient blood pressure drop did coincide with a brief slowing of the heart, and was probably caused by vagal stimulation set up by tugging on the lung root. Vagal arrest never occurred.

The adjustment of the respiratory system to the sudden removal of one functioning lung was complete in that oxygen continued to be absorbed at a constant rate. It is obvious, then, that the remaining lung began immediately to absorb as much as double the oxygen it had absorbed prior to ligation.

The changes in ventilation of the remaining lung were unpredictable. Theoretically, oxygen consumption could be increased by either increasing the minute volume of respiration or the efficiency of oxygen absorption or both.

In over one-half the dogs the recorded minute volume did not change, which means, in actual fact, that in these cases the minute volume of the one remaining lung approximately doubled. Two factors determine minute volume: rate of respiration and tidal air. Over-all minute volume was maintained unchanged either by a large increase in tidal air of the remaining lung or by a smaller increase in tidal air together with an increase in respiratory rate.

In other dogs there was altered efficiency of oxygen absorption, as measured by the ventilation equivalent, due to changes in the minute volume after liga-

tion and no real change in oxygen use. Since the ventilation equivalent is a ratio between the minute volume and the oxygen uptake, it varies as these two factors vary. The rate and depth of respiration are controlled by complex nervous and chemical mechanisms. Oxygen absorption in the lungs, on the other hand, is a physical process depending upon such conditions as the permeability of the alveolar and capillary membranes, the area of the absorbing surfaces, the time of exposure of the blood to the alveolar air, the total volume of blood exposed to the alveolar air in any period of time, and the difference in partial pressure of the oxygen of the alveolar air and the blood of the pulmonary artery. Minute volume and oxygen consumption may vary independently, and the ventilation equivalent may be affected by alterations in either. For example, in Dog 18 the ventilation equivalent before ligation was 3.9, for 30 seconds after ligation the tidal air was very small and the rate of respiration slow, but oxygen use was not reduced proportionally to minute volume. The ventilation equivalent at this time was 1.8. Respiration gradually became deeper and faster; oxygen consumption returned to the preligation level and the ventilation equivalent became 2.9. These changes in ventilation equivalent denoting altered efficiency of oxygen absorption per liter of ventilated air depend upon reduction of minute volume of respiration rather than changes in oxygen uptake following ligation. Oxygen absorption was but little affected by the operative manipulation, whereas respiratory movements were momentarily inhibited, presumably through nervous reflex mechanisms.

Since the ventilation equivalent which relates the two factors of minute volume and oxygen uptake is not a constant, it does not appear to be a necessarily reliable index of respiratory function, at least when conditions are basal. There is, of course, a limit to the capacity of a lung to absorb oxygen. This limit may be reached more quickly in lungs with diseased alveolar membranes or greatly decreased alveolar surfaces. To approach such a limit would mean increasing the oxygen demand until the lowest possible ventilation equivalent had been attained. Under such conditions of stress the ventilation equivalent might prove to be a better criterion of cardiorespiratory function.

These studies do not indicate to how much additional stress, such as exercise or an increased blood volume, an animal could adjust after pneumonectomy. Longacre and associates[15, 16] found some indication of the loss of cardiorespiratory reserve after pneumonectomy in chronic experiments measuring responses of dogs to exercise, using pulse, respiratory rate, body temperature rise, and arterial oxygen saturation as criteria of function.

Ligation of one lung root does put increased stress on the right ventricle. Right intraventricular pressure increased in all but one of the cases measured. Fineberg and Wiggers[6] have found that the right ventricle, like the left, is able to meet increased peripheral resistance by an increased force of contraction, but that the range of compensation is extremely narrow. Almost all of our dogs were able to adjust to the added stress put on the right ventricle. Although 40 per cent showed an immediate reduction in cardiac output, systemic blood pressure was maintained. What happened to the cardiac output in the

succeeding hours was not determined. Tying off the right branch of the pulmonary artery is equivalent to occlusion of at least 50 per cent of the pulmonary artery. Fineberg and Wiggers found one dog in their series which was unable to maintain right ventricular output unchanged when 41 per cent of the pulmonary artery was occluded. One dog in our series with apparently normal heart and lungs was unable to maintain the systemic blood pressure after sudden pneumonectomy.

With the pulmonary vascular bed cut in half, it might be expected that the pressure in the pulmonary circuit would be increased more readily. Gibbon, Gibbon and Kraul[17] showed that in cats when the right middle and lower lobes and all of the left lower lobes were removed a blood transfusion of 15 c.c. per kg. caused death due to pulmonary edema in four out of nine animals. A similar type of study before and after pneumonectomy might prove enlightening.

In conclusion, the majority of dogs in this series were able to make physiologic adjustments to the functional loss of one lung; two dogs, one with heart disease and one with apparently normal heart and lungs, were not. No attempts were made to measure the cardiorespiratory reserves after such a procedure.

SUMMARY

1. The details of cardiovascular and respiratory adjustment to functional pneumonectomy were studied in twenty dogs with closed chests. Pneumonectory was accomplished under barbiturate anesthesia by the sudden ligation of one entire lung root.

2. The immediate response of the cardiovascular system was a rise in right intraventricular pressure, and, in 40 per cent of the dogs, a decrease in cardiac output. Systemic blood pressure was usually maintained.

3. Oxygen consumption before and after ligation was essentially unchanged.

4. The changes in pulmonary ventilation were unpredictable and consisted of increases in tidal air of the remaining lung, increases in rate of respiration, and altered efficiency of oxygen absorption.

5. Two dogs were unable to compensate for this functional pneumonectomy. One showed cardiac disease, and one had apparently normal heart and lungs.

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